The DIGESTIVE TRACT *in*

ROENTGENOLOGY

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MY WIFE ESTELLE

whose constant encouragement and co operation have been of inestimable value in the preparation of this work

Preface

The diagnostic value of the roentgenographic method in the study of the digestive tract is fully recognized by internist and surgeon alike A knowledge of its potentialities should be of great value to the general practitioner

For more than a quarter of a century it has been my privilege to be affiliated with Bellevue Hospital During this period I have had the opportunity of correlating the roentgenographic findings in disorders of the digestive tract with those obtained at operation or autopsy This book is an outgrowth of my experience with the enormous amount of material at the hospital and of my own private practice

In the pages of this book the reader will find a presentation of those technical procedures which I have found most helpful and of the abnormalities of the digestive tract both functional and organic in the study of which this diagnostic aid is of inestimable value

Some of the illustrations in this volume appeared in my earlier volume Clinical Roentgenology of the Alimentary Tract, published by W B Saunders Company and I give them my thanks for permission to reproduce these illustrations

I wish to express my gratitude for the excellent co-operation that I have received from the various members of the hospital staff and to the publishers J B Lippincott Company for the many courtesies extended to me

I wish in particular to record my debt to Dr Lewis Gregory Cole whose inspiring lectures were my first introduction to the field of roentgenology of the digestive tract when I was a medical student at Cornell to Dr Russel D Carman whose book I read and re read while a medical officer during World War I to Dr I Seth Hirsch formerly Director of the Roentgen Department of Bellevue Hospital to Dr Lewis J Friedman the present director of the department to Dr Eugene F DuBois and Dr David P Barr of the Cornell Medical College and to Dr Louis Hauswirth Director of Medicine Beth David Hospital

JACOB BUCKSTEIN

Contents

INTRODUCTION

THE HYPOPHARYNX AND ESOPHAGUS

THE STOMACH

THE DUODENUM

THE SMALL INTESTINE

THE LARGE INTESTINE

HERNIATION AND EVENTRATION OF THE DIAPHRAGM

THE GALLBLADDER AND BILE DUCTS

THE SPLEEN, LIVER AND PANCREAS

INTRODUCTION

1

History of Roentgenology of the Alimentary Tract

It was on December 28 1895 that the modest and retiring Wilhelm Konrad Rontgen, Professor of Physics at the University of Wurzburg reported his experience with a strange phenomenon that was destined to revolutionize the world The work was reported in the form of a preliminary paper entitled On a New Kind of Rays and published in the Annals of the Physical Medical Society of Wurzburg Within a short time Rontgen s original article was translated into all the important languages of the world and it became the universal property of mankind

In this paper he wrote

Of special significance in many respects is the fact that photographic dry plates are sensitive to the x rays wherever it has been possible I have controlled by means of photography every important observation which I have made with the eye by means of the fluorescent screen

In these experiments the property of the rays to pass almost unhindered through thin sheets of wood paper and tinfoil is most important The photographic impressions can be obtained in a non darkened room with the photographic plates either in the holders or wrapped up in paper On the other hand from this property it results as a consequence that undeveloped plates cannot be left for a long time in the neighborhood of the discharge tube, if they are protected merely by the usual covering of pasteboard and paper

The retina of the eye is not sensitive to these rays Even if the eye is brought close to the discharge tube, it observes nothing although as experiment has proved the media contained in the eye must be sufficiently transparent to transmit the rays

The justification for calling by the name rays the agent which proceeds from the wall of the discharge apparatus I derive in part from the entirely regular formation of shadows which are seen when more or less transparent bodies are brought between the apparatus and the fluorescent screen (or the photographic plate)

I have observed and in part photographed many shadow pictures of this kind the production of which has a particular charm I possess for instance photographs of the shadow of the profile of a door which separates the rooms in which on one side, the discharge apparatus was placed on the other the photographic plate the shadow of the bones of the hand the shadow of a covered wire wrapped on a wooden spool of a set of weights enclosed in a box of a compass in which the magnetic needle is entirely enclosed by metal of a piece of metal whose lack of homogeneity becomes noticeable by means of the x rays etc

One may well imagine the astounding effect upon the world of this miracle ' by which hidden areas within the human body could be made visible to the naked eye

Fluoroscopy so essential a procedure in the roentgenology of the alimentary canal was first discovered when Rontgen saw the effect of the rays generated by his Hittorf Crookes tube on a barium platino cyanide screen In this first paper he wrote

If the hand be held between the discharge tube and the screen the darker shadow of the bones is seen within the slightly dark shadow image of the hand itself

There then followed a gradual development in the perfecting of the modern fluoroscope One of the earliest fluoroscopes described in this country was that of an American engineer E P Thompson

Haschek and Lindenthal[1] in January, 1896, injected the hand of a dead body, by way of the brachial artery, with Teich

mann's "mass," which consisted primarily of chalk and should therefore be as opaque *to the roentgen rays as the bones* After an exposure of 57 minutes, they were able to show the blood vessels in the roentgenogram even more distinctly than the bones themselves In a second case they roentgenographed the *little finger of the left* hand of a colleague, this had been injured by a bullet and showed the presence of callus formation They recognized in this finding a new method for the demonstration of bone pathology The actual roentgenograms accompany the article They wrote 'Should it be possible through technical improvement to radiograph other portions of the body, this would open up a new field in diagnosis for the healing of the sick'

An important step forward occurred with the publication of the highly interesting paper of Sehrwald The importance of his contribution rested on the fact that he showed that chlorine, bromine and iodine absorbed the roentgen rays and that this property depended upon the atomic weight of the elements He predicted that internal organs filled with these halogens would become visible to the roentgen rays

The next step in the development of the potentialities of the roentgen method was in the visualization of those internal structures of the human being, such as the esophagus stomach and the rest of the digestive canal This occurred with the publication of the work of Becher[3] He recorded his observations on the outline of the stomach and intestines of a guinea pig *after first filling them with lead acetate* He hit upon the two great essentials on which further progress depended, namely a solution must be employed which can be introduced into the human stomach without causing damage and which is non transparent for roentgen rays

Within a short time Wegele[4] had made a suggestion which was later carried out by Lindemann,[5] who first placed a copper wire into a rubber tube, which he then introduced into the stomach of a human being he succeeded in roentgenographing this, *outlining the curvature of the stomach* He also succeeded in doing this in the case of a patient with gastric dilatation Actual roentgenograms which required a 16 minute exposure accompany his interesting account

Less *than a month after the original contribution* of Becher, Hemmeter[6] published his paper on the roentgen visualization of the human stomach In his original article Hemmeter, after describing the attempt of *Becher to roentgenograph the stomach and* a loop of intestine of a guinea pig, said "To obtain a photograph of the human stomach a solution is necessary having two properties (1) it must not injure the stomach of the subject to be photographed (2) it must be impenetrable to the Rontgen rays" He did say, however, 'I do not consider the use of the Rontgen method on account of its complexity and long duration of exposure, as practical for determining the size and location of the stomach" While suggesting that lead acetate might be put into an elastic bag introduced into the stomach and then roentgenographed, he personally did not put the procedure to practical effect

Rumpel[7] then outlined a dilated esophagus by filling it with a suspension of bismuth subnitrate

The pioneer work of Cannon in the early development of the roentgenology of the alimentary tract will forever remain a credit to American science Because of its historic significance I quote at length from his paper, published in 1914, on the "Early Use of the Roentgen Ray in the Study of the Alimentary Canal

In the fall of 1896, when I was a first year student in the Harvard Medical School and Mr Albert Moser a Cambridge acquaintance was a second year student we went to Professor H P Bowditch and asked for an opportunity to undertake a physiologic investigation He suggested that we test by means of the newly discovered Roentgen rays the Kronecker Meltzer view of deglutition namely, that substances when swallowed are

shot down the oesophagus by pressure developed in the mouth and are not pushed down by a peristaltic wave A small static machine and some simple tubes were secured, and we set to work Our first observation was made on Dec 9 1896 when we watched globular pearl buttons pass down the oesophagus of a dog On Dec 14, we repeated the observations on a rooster whose neck was kept straight by fastening the head and body in fixed positions

That substances vary in their opacity to the Roentgen ray in accordance with their molecular weight was then common knowledge Barium and bismuth were selected from among the heavy metals and barium sulphate and bismuth subnitrate were chosen as salts which because practically insoluble would serve our purposes Since bismuth subnitrate was a pharmacopeial preparation we determined first to use that[8]

On Dec 16 1896 we gave a frog a gelatin capsule filled with this bismuth salt and after seeing clearly the shadow of the substance in the frog s stomach we repeated the next day Dec 17 the use of the salt in a capsule to show deglutition in the dog When the swallowed capsules were dissolved the bismuth subnitrate was liberated and in my notes I find the remark that thus a dark roundish area was produced as a stomach shadow Then we procured a goose and made for it a box so arranged that the long neck reached up through the cover A high cardboard collar was then attached to the top of the box in such a way that it could be closed in front when surrounding the goose s neck Thus the goose with the appearance of using the most stylish neckwear presented to the fluorescent screen a very satisfactory extent of oesophagus At a meeting of the American Physiological Society in Boston Dec 29 1896 the phenomenon of deglutition as exhibited by the goose when swallowing capsules containing bismuth subnitrate was informally demonstrated to the members by means of the Roentgen rays This was I think the first public demonstration of movements of the alimentary tract by use of the new method Later I showed that barium sulphate mixed with the food was quite as satisfactory as bismuth subnitrate[8]

It was not until Jan 9 1897 that we employed bismuth subnitrate mixed with food (in this case a bread mush) to render the swallowed mass visible And in order to test the rate at which substances with different consistencies move along the oesophagus we added more or less water to the mush thus varying its physical state from a rather stiff mass to a very soft nearly fluid mixture Various observations of this nature were made on the goose in January February and March 1897 On April 3 1897, we began using the same mush bismuth bolus and also meat with bismuth subnitrate adherent in a study of swallowing in the cat and as the mixture of bismuth subnitrate and food accumulated in the stomach that organ was outlined by a dark shadow On April 3 and in observations on several days thereafter the gastric contents became visible as the cats swallowed more and more of the mixture but no movements of the wall were seen Finally on April 23 1897, a cat was fed bread soaked in warm water and mixed with bismuth subnitrate about an hour and a half later peristaltic waves were clearly seen passing over the organ

At a meeting of the American Physiological Society in Washington May 4 5 and 6 1897 a preliminary report of these observations was presented by Professor Bowditch and the summary of the report was published a month later (Science June 11 1898 p 901) A part of the summary was as follows

' For this purpose (studying the oesophageal and gastric movements by means of Roentgen ray shadows) moist bread meat mush or viscid fluids were mixed with bismuth subnitrate Food thus prepared is visible during the process of deglutition and if given in sufficient quantities serves to outline the stomach and to render its peristaltic movements visible Then followed an account of the results which had been obtained in observing deglutition in different animals and also gastric peristalsis

Roux and Balthazard[9] in June 1897 described a series of experiments on the motor function of the stomach of various animals by means of the roentgen ray by employing a mixture containing bismuth subnitrate In July of that year they gave a brief report of their experiences with the method in studying the contractions of the stomach of man In a later publication they[10] continued their studies on the movements of the stomach by the roentgen method with the administration of bismuth subnitrate and presented actual radiographs They concluded their study by saying

The stomach is divided into two distinct regions the largest part of the stomach serves

as a reservoir for food the prepyloric portion is the motor region of the stomach, and by its vigorous peristaltic movements it forces into the duodenum little by little, the material accumulated in the stomach

In 1898, Cannon[11] published the results of his research on the behavior of the esophagus, during which experiments he recorded his findings in the case of a girl of 7 years to whom he administered a suspension of bismuth in water That same year and in the following year, 1899, Cannon worked with Dr Francis H Williams in the roentgen visualization of the stomach of a boy aged 10 and that of his 7 year old sister They were given a meal of a pint of milk and bread to which was added 30 Gm *of bismuth subnitrate After the* outline of the stomach was visualized on the fluorescent screen, drawings were made on transparent paper placed over the gastric image These results were published in 1903[1]

Rieder[13] in 1905 recommended a mixture of flour or potato with bismuth subnitrate for the visualization of the alimentary tract He recommended the use of 30 Gm of bismuth by mouth

Of historical interest is the original contribution of Holzknecht and Brauner[14] They presented a detailed description of the technic of fluoroscopic study of the bismuth filled stomach In some cases they not only administered a suspension of bismuth but introduced in addition an effervescent mixture to produce gaseous distention of the stomach Included in this contribution are drawings of the appearance of the stomach under different conditions Of interest also is a photographic reproduction of the laboratory showing a picture of Holzknecht and a number of other doctors in front of the fluoroscopic screen observing a patient who is being examined

Holzknecht and Jonas carried roentgen exploration still further in their contribution of 1909[15] They described their combined fluoroscopic and roentgen procedure in the examination of the stomach and duodenum and stressed the importance of palpatory manipulation The patient, placed behind the screen with his stomach empty, was examined following the administration of 10 to 15 Gm of bismuth carbonate in 100 cc of water The passage of the opaque suspension was followed through the esophagus into the stomach and was then forced manually into the pylorus and duodenum They studied the form position and peristalsis of the stomach and emphasized the normal nature of the air in the stomach, the presence of which in the pre roentgen era had been attributed to nervous phenomena They emphasized the significance of fluoroscopy in the study of the anatomy and physiology *of the normal stomach and stressed its* diagnostic value in the demonstration of nonpalpable tumors, pyloric stenosis, carcinoma, adhesions and malfunction of a gastroenteric stoma

So remarkable were the potentialities of this new method of diagnosis that H Beclere,[16] envisioning the possibilities of the future, said

> The introduction into the stomach of a very large quantity of bismuth in a mixture of bread and milk has enabled Roux and Balthazard in France and Williams and Cannon in America to make interesting researches in animals children and adults on the muscular function of the digestive canal It is to be hoped that clinical medicine will take part in these researches and that in this manner visceral radiology will some day be enriched *by a new chapter*

That the hope of Beclere has met with rich fruition is demonstrated by the accuracy which the roentgen method has achieved and the reliance placed by the clinician and surgeon upon the objective significance of the information which is obtained

In the following pages of this book will be unfolded the story of the achievements in diagnostic roentgenology of the alimentary tract, which have fully justified the prophecy of Beclere Much of this has depended on the elaboration of technical aids

in the construction of apparatus, and an incalculable debt is due the physicists and engineers who have contributed to this progress No attempt will be made in this work however, to discuss details regarding the structure of roentgen apparatus tubes, cassettes super speed screens or any of those other features which can best be learned from actual contact with such apparatus or from books specifically devoted to the subject of technic or in a course in the physics of roentgenology—best learned in conjunction with the subject of general roentgenology itself

Such details as concern the preparation of the patient the nature of the mixture recommended for administration to the patient the position of the patient the time interval between exposures and similar points of a clinical character however will all be discussed in relation to the specific phase of the alimentary tract under investigation—each in its appropriate chapter

One feature of a technical character which I do wish to emphasize is the use of a Bucky diaphragm attached to the fluoroscopic screen I consider this added feature of inestimable value in affording a clearer and more detailed visualization of the image, and I strongly recommend that it be made a permanent addition to the fluoroscopic screen in the study of the alimentary canal

The modern fluoroscope is constructed in such a manner as to give a maximum degree of safety in diagnostic exploration Various spot film devices are now obtainable for graded compression of an isolated area of the alimentary tract, with a switch over for prompt radiographic exposure of the region under special observation If in addition to the safety devices constructed within the fluoroscope itself for the protection of the operator a chair is employed with a lead protective apron in front, if soft lead lined gloves are used and if intermittent exposure is made of as small an image as is consistent with proper visualization of the various portions of the viscus being studied, the procedure may be considered essentially safe

At present, the question as to whether one should depend on fluoroscopy or upon roentgenography in the examination may be considered obsolete The combination of both procedures fluoroscopy and roentgenography is essential in order to obtain the maximum of detail of a diagnostic nature in the roentgen examination of the alimentary tract Both procedures complement each other and there is peril in the entire elimination of one or the other Certainly fluoroscopy will give extremely valuable information regarding the grosser details of structure and the kaleidoscopic changes of physiologic behavior Many lesions even though small will be picked up by the expert on fluoroscopic examination alone, if the patient is thoroughly examined through every angle and if manual compression is employed to bring out the mucosal relief For the finer changes of structural detail the radiograph has its advantages Maximum safety in diagnosis consists therefore in the employment of both procedures in every case

Moreover, the roentgenogram serves as a permanent record of the findings and is free of the personal equation involved in a drawing of an evanescent fluoroscopic image Such an objective record as the roentgenogram can be studied at great length, in leisurely fashion at any time and long after the fluoroscopic observation has been completed It may serve for teaching purposes and may be placed in a viewing box in the operating theater as a guide in cases of surgical intervention A record of this kind can also be used for purposes of comparative study in the determination of the effectiveness of medical management of any particular disorder and as in the pages of this book, such roentgenograms can be used for purposes of publication No matter how skillfully trained the gastro enterologist or radiologist may be there are obvious advantages in the correlation of both fluoroscopy and roentgenography in the examina

tion of any patient in whom a lesion of the gastro intestinal tract is suspected

Finally, I wish to stress the importance of the clinical background of every such case that is studied radiographically While the objective roentgen findings in many cases are of such a clear cut character that their diagnostic significance may be accepted unequivocally, there are not infrequently borderline cases in which only the interplay of all the factors that can be brought to bear, whether clinical, pathologic or radiologic, will enable us to render maximum diagnostic service to our patients

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THE HYPOPHARYNX
AND
ESOPHAGUS

2

The Hypopharynx

ROENTGEN EXAMINATION OF THE HYPOPHARYNX

The act of swallowing is a complicated reflex initiated by a chain of physiologic factors in the pharyngeal region The pharynx is a musculo membranous organ about 5 inches in length which extends from the base of the skull to about the level of the sixth cervical vertebra where it becomes continuous with the esophagus By virtue of its communications anteriorly it is divided into a nasal pharynx, an oral pharynx and a laryngeal pharynx The laryngopharynx extends from the laryngeal opening to the cricopharyngeal constrictor It is thus bounded above by the epiglottis below which is the superior aperture of the larynx Laterally are the pyriform sinuses Medial to the sinuses are the aryepiglottic folds while laterally are the thyroid cartilage and the hypothyroid ligament Above the epiglottis are two other recesses, the valleculae The pharyngoepiglottic folds are at the lateral margins of the valleculae and extend from the epiglottis to the pharynx

The bolus of masticated food is passed through the isthmus of the fauces by the action of the anterior portion of the tongue against the palate This is also aided by the posterior portion of the tongue forcing the bolus through the pharynx into the esophagus[1] The bolus is passed through the pharynx into the esophagus in about one fifth of a second from the time of contraction of the mylohyoid muscles This complicated act is controlled by a center in the medulla with the effectors present in the muscles of the larynx and the pharynx During this process the oral and nasal cavities are closed off and the larynx is occluded by the adduction of the vocal cords and the elevation of the larynx When because of any disorder there is interference with the elevation of the larynx swallowing is impossible

The junction of the hypopharynx and the esophagus was described by Killian in 1908 on the basis of endoscopic studies He described a transverse fold on the posterior wall of the esophagus at its junction with the hypopharynx Ordinarily this fold occluded the opening of the esophagus but this region relaxed on swallowing and when a vomiting reflex was produced He called this area the ' mouth of the esophagus and called the bulge on the posterior wall the esophageal lip He correlated the anatomic localization of the lip with the transverse portion of the cricopharyngeus muscle

That the indrawing of the posterior wall of the pharyngoesophageal junction is due to the activity of the transverse portion of the cricopharyngeus muscle is supported by the following observations

1 In favor of the assumption that this smooth indentation is due to the functional behavior of the cricopharyngeus muscle is the fact that pulsion diverticula originate in this region It is a generally recognized anatomic fact that these diverticula originate on the posterior wall of the pharyngoesophageal junction in the weak area between the oblique and transverse fibers of the cricopharyngeus muscle Since the smooth indentation on the posterior wall corresponds to the area just below the origin of these diverticula, there is added confirmation that the concavity is produced

by the action of the transverse portion of this muscle

2 In studying the roentgen appearance of this concave indentation one may note that there is considerable variability in the degree of constriction in this region and in the length of the segment involved At times, this area may be completely shut off, as in marked dilatation of the esophagus in cardiospasm or even in carcinoma, thereby preventing regurgitation into the pharynx and ultimately into the tracheobronchial tree One may best explain such a phenomenon as being due to constriction produced by a muscular structure capable of alteration in its physiologic behavior, now yielding and now causing occulsion to a variable degree The anatomic localization of this indentation, as well as its behavior and its association with the origin of the classical pulsion diverticulum in this region, all point strongly to the transverse portion of the cricopharyngeus muscle as the cause

ROENTGEN EXAMINATION OF THE HYPOPHARYNX

While the pharynx and the pharyngo esophageal junction may sometimes be noted after a simple swallow of barium, the passage of the opaque fluid is often so rapid as to make careful visualization of this region difficult or impossible One may caution the patient not to repeat the swallowing act after the first mouthful of barium has filled out the pharynx thus permitting some of the barium to outline the contour of the pharynx as well as the valleculae and the pyriform sinuses An aid in arresting the barium for a more protracted period of observation is the Valsalva test This may be carried out as follows The patient is told to take a deep breath Before expiration he keeps his mouth closed tight and his nose is pinched He is then instructed to exhale As a result, the increased intrathoracic pressure transmitted by way of the trachea distends the pharynx with air In this manner, the barium within the recesses of the pharynx may not only be retained but will stand out in sharp contrast In many cases, however, the Valsalva method is ineffectual in delaying the rapid emptying of the pharynx

Retention of barium in the pharynx and in the pharyngeal recesses will in part depend on the nature of the mixture, since a thick paste will obviously cling to these regions more readily than a simple barium water suspension The method of preparing the thick mixture will be described in connection with the roentgen examination of the esophagus The visualization of the pharyngeal structures will also depend on the prompt fixation in the roentgenogram at the optimum moment of exposure, usually after the first swallow of a small amount of the thick mixture and before subsequent swallowing has dislodged the barium Spot film technic is an invaluable aid Examination in the supine position may also be helpful in retarding the flow of barium In some cases there may be retention of barium in the recesses without either subjective complaints or evidence of organic derangement as shown by esophagoscopic examination *In other cases, however,* with such retention, particularly when the quantity is marked and the period prolonged actual organic changes may be noted some of which are recognizable on roentgen examination

As viewed laterally the wall of the hypopharynx when distended with barium may show a slight superficial irregularity of its anterior surface The posterior wall is smooth and often exhibits wavelike ripples apparently due to peristaltic activity The more generally recognized alteration is on the posterior wall at its junction with the esophagus and appears as a smooth rounded indentation the anatomic basis for which has already been described The narrowing at the junction of the pharynx and the esophagus may at times produce practically complete occlusion because of the functional behavior of the cricopharyngeus muscle This may act as a protective mechanism by preventing regurgitation into

the pharynx and trachea in the presence of an obstructive lesion of the esophagus of either organic or functional origin

Viewed anteriorly, the pharynx when distended with barium is of smooth contour usually tapering gradually at its junction with the esophagus After partial emptying of the pharynx, rests of barium

region is studied however the fewer are the cases of dysphagia of a purely functional nature The most serious cause of intrinsic origin is carcinoma of the hypopharynx Foreign bodies caught at the mouth of the esophagus may similarly cause obstruction A pulsion diverticulum when large may compress the pharyngo-esopha





Fig 1 (*Left*) Showing the appearance of the hypopharynx as viewed anteriorly with barium outlining the recesses Note the valleculae above and the pyriform sinuses below

Fig 2 (*Right*) Normal hypopharynx (right lateral view) Note the smooth posterior wall and the superficial irregularity of the anterior wall

may be noted outlining the valleculae which appear as two small cup shaped areas to either side at about the level of the thyroid cartilage Below are the bilaterally placed, triangular shaped pyriform sinuses In the lateral view one or another of these structures may be seen but often only with difficulty or not at all, becau e they may overlap each other

Abnormalities of the hypopharynx may result from a number of causes both functional and organic The more carefully this

geal junction This will be discussed more fully in the chapter on diverticula of the esophagus

A comparatively rare but clinically important disorder is the dysphagia occurring mainly in middle aged women and associated with secondary anemia a condition which had originally been considered hysterical and is occasionally still referred to as globus hystericus The association of anemia with this condition was emphasized by Blankenstein[3] in 1893 in whose disser

tation will be found references to the older literature on the subject Plummer in 1914 and Vinson in 1922 thought that the dysphagia was purely functional, although Paterson[4] in 1906 had described atrophic changes in the pharynx as a concomitant finding in this syndrome

eaten unusually fast, the large webs may be a cause of definite obstruction and may be the underlying organic process in individuals stamped with the diagnosis of "globus hystericus"

The presence of organic changes in the hypopharynx and the upper esophagus in

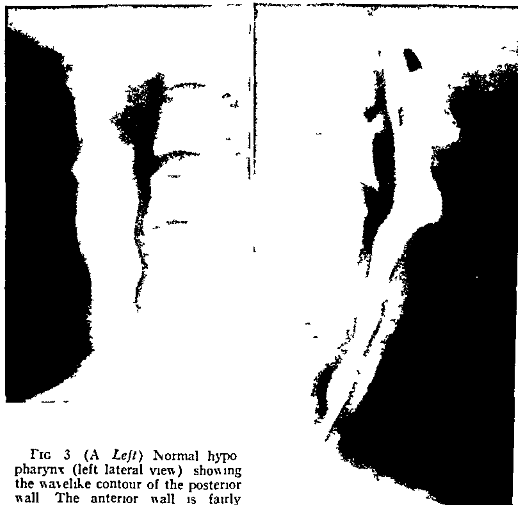


Fig 3 (A *Left*) Normal hypopharynx (left lateral view) showing the wavelike contour of the posterior wall The anterior wall is fairly smooth
(B *Right*) Normal hypopharynx (right lateral view) Note the wavelike contour of the posterior wall and the fairly smooth anterior wall

That organic changes of the pharyngoesophageal region are present in this disorder was also shown by Mosher[5] who described the presence of webs at the entrance to the esophagus behind the cricoid cartilage They appear as thin folds of mucous membrane and scar tissue roughly crescentic While the small web may be present without symptoms unless food is

association with the syndrome of dysphagia and anemia has been corroborated by a number of observers Kelly[6] Paterson[7] in his later article, Suzman[8] and Hoover[9] One of the cases of dysphagia and anemia reported by McGee and Goodwin[10] was studied at autopsy The upper portion of the esophagus was hyperemic and showed irregular areas of denudation with islands

of lymphocytes and scattered plasma cells The muscular wall was thickened evidence of a chronic esophagitis

There is, therefore ample evidence of an underlying organic basis for this type of dysphagia associated with anemia

Of great significance is the apparent predisposition of these organic alterations to the ultimate development of carcinoma of the hypopharynx (Paterson,[7] Ahlbom[11] and Simpson[1])

Many of these changes may be recognized on radiologic examination of the hypo pharynx (Mosher[5] and Waldstrom and Kjellberg[13]) Webs appear as smooth nar row indrawings of the contour present mainly on the anterior wall of the hypo pharynx and directed obliquely downward There may be one or more and they are best seen when the hypopharynx is well dis tended with barium and with the patient in the lateral position In advanced cases these incisurae may also be noted in the antero posterior view In the very chronic cases infiltration of the wall may produce a stricture distinguishable only with diffi culty from a malignant lesion

In other cases the narrowing and irregu larity produced by actual malignant infil tration of the hypopharynx may be recognized on roentgen examination

Marked retention of barium may be noted in the valleculae and the pyriform sinuses in the dysphagia of myasthenia gravis (Veits[14])

Figure 1 shows the appearance of the hypopharynx as viewed anteriorly with barium outlining the recesses Note the valleculae above, the pyriform sinuses be low, and the barium finally outlining the folds in the proximal portion of the esophagus

Viewed laterally (Fig 2), the anterior wall is superficially irregular The posterior wall, on the other hand, is smooth and sharply defined

At times, however (Figs 3A and 3B) the anterior wall may be fairly smooth The posterior wall instead of being a straight line may be wavelike possibly as a result of peristaltic activity

Not uncommonly near the distal end of the posterior wall of the hypopharynx one may note a smooth indentation which represents the constriction produced by the transverse belly of the cricopharyngeus muscle

The smooth indentation with constriction produced by the cricopharyngeus muscle is



Fig 4 The constriction produced by the transverse portion of the crico pharyngeus muscle is particularly well shown in the above illustration

particularly well shown in Figure 4 Note the distention of the pharynx with air prox imal to this constriction and the beginning of the esophagus distal to it

A marked exaggeration in the degree of constriction at the junction of the hypo pharynx and the esophagus may give rise to dysphagia

Illustrative Case I G male aged 62 The patient a known cardiac was admitted to the hospital complaining of inability to swallow even fluids and of vomiting of 3 days duration

Three years previously he had had a simi lar episode of dysphagia He had lost 45 pounds in the preceding 4 years Physical ex



FIG 5 Functional obstruction at the pharyngo esophageal junction



FIG 6 Obstruction of the cricopharyngeal region by a prune pit

amination revealed evidence of auricular fibrillation His hemoglobin was 90 R B C 5,200 000

Roentgen examination at this time (Fig 5) revealed obstruction in the pharyngo esophageal region with reflux of the barium by way of the pharynx into the trachea The evidence was that of an obstructive lesion in that region

On esophagoscopy the instrument was passed with ease into the stomach No obstruction or foreign body was seen nor was any other explanation of the symptoms found Neurologic examination was negative

After 4 days the patient s dysphagia suddenly disappeared and he was able to drink liquids and even eat solid foods Nine months later the patient returned because of dimness of vision and colored scotomata during the preceding 3 to 4 days His cardiac symptoms were well controlled by digitalis He had no dysphagia at this time

The fact that he had had two episodes of dysphagia 3 years apart the negative findings on esophagoscopy the short duration and abrupt termination of the second attack with complete relief of the dysphagia thereafter all point strongly to a functional derangement as the cause probably in the nature of an extreme degree of cricopharyngeal spasm with total constriction of the lumen in this region

Obstruction of the cricopharyngeal region by a prune pit is shown in Figure 6 The narrowing of this area by the action of the transverse portion of the cricopharyngeus muscle probably contributed to the arrest of the prune pit in this region

Malignant infiltration of the hypopharynx is illustrated by the next case

A M female aged 69 During the preceding 3 months the patient had been complaining of progressive dysphagia at first with solid food only but at the time of her admission to the hospital with liquids as well She had lost considerable weight

Endoscopy disclosed a mass which bled easily A biopsy was taken

The pathologic report was squamous cell carcinoma

The roentgen examination (Fig 7) showed an irregular tubular narrowing of the hypopharynx which was interpreted as representing an infiltrating lesion in this region It is interesting to note that there had been no history of difficulty in swallowing prior to the onset of the brief period of the dysphagia which brought the patient to the hospital There was no clinical evidence to indicate that the carcinoma was etiologically related to any preceding underlying pathology of the hypopharynx

The roentgen findings in tumor of the hypopharyngeal region may be noted in the following case

G H, male, aged 63 During the preceding 2 months the patient stated that he had had difficulty in swallowing and had had a



Fig 7 Carcinoma of the hypopharynx Note the narrowing and distortion of the hypopharynx and the superficial irregularity of the anterior wall

chronic cough When he attempted to swallow food some of it regurgitated back through his nose He was very hoarse Physical examination disclosed a firm fixed mass at the lateral end of the hyoid bone on the left side anterior to the sterno cleido mastoid muscle and attached to the deeper structures and not to the skin The mass measured 4 x 3 cm, was not tender or cystic There was a smaller mass above this which was also fixed but more movable than the larger mass and seemed to be a node There was a firm nodular mass attached to the left palatophar



Fig 5 Functional obstruction at the pharyngo esophageal junction



Fig 6 Obstruction of the cricopharyngeal region by a prune pit

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yngeal fold, which appeared to extend posteriorly as well as anteriorly and to push the fold forward

Laryngoscopic examination disclosed a large ulcerating mass in the left postpharyngeal wall, measuring about 3 x 2 cm, with a large crater in the center The mass appeared to extend along the lateral pharyngeal wall to the posttonsillar pillar and the base





Fig 8 (A *Left*) Carcinoma in the left lateral wall of the hypopharynx Note the obliteration of the left pyriform sinus

(B *Right*) Carcinoma of the hypopharynx Note the irregularly outlined translucent areas produced by the tumor

of the tongue A biopsy taken from the lateral pharyngeal wall showed squamous cell carcinoma

Roentgen examination (Fig 8A) revealed a defect of the left lateral wall of the hypopharynx with displacement to the right and an obliteration of the left pyriform sinus

The diagnosis was tumor of the left hypopharyngeal region

A carcinoma of the hypopharynx may be characterized by the presence of translucent areas produced by the tumor

C D female age 62 The patient gave a 1 month history of difficulty in swallowing and of the feeling of a lump in her throat She was able to swallow only small amounts of liquid At times the liquid would be forced out through her nose On one occasion she brought up a small amount of fresh blood

"Using a number 16 laryngoscope the pharynx was explored and almost immediately a large fungoid mass was encountered involving part of the right and almost all of the left arytenoid This mass extended downward and incorporated the entire central hypopharynx and cricopharyngeus muscle which could not be dilated with the smallest dilator From this mass a biopsy was obtained

The diagnosis on pathologic examination was squamous cell carcinoma

Roentgen examination (Fig 8B) shows a number of irregularly outlined translucent areas throughout the region of the hypopharynx produced by the tumor

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produced by the left bronchus may be encountered

Other areas of the esophagus may appear constricted because of its somewhat twisted course Pratje[4] explained in this manner an apparent constriction in the lower end of the esophagus a few centimeters above the diaphragm Under such circumstances the esophagoscopist may note an abrupt closure of the lumen which he may misinterpret as being due to an actual area of constriction

Below the impression on the esophagus produced by the aortic arch one may sometimes note the effect upon it of the pressure produced by the left bronchus In order to determine the relation of the right pulmonary artery to the left bronchus and the esophagus Evans made a careful dissection of the posterior mediastinum in three cadavers He found that neither the pulmonary artery stem nor its branches were in direct apposition to the esophagus The pulmonary artery stem divided in front of the left bronchus The right pulmonary artery was separated from the esophagus by a space filled with connective tissue and lymph nodes He also impregnated the dissected specimens with barium and made roentgenograms showing that the impression on the esophagus below the aortic arch was produced by the left bronchus This impression by the left bronchus may be secondarily influenced however by the pressure of an abnormal right pulmonary artery

At times it may be possible to see three backward curves from above downward (1) the aortic impression (2) the left bronchus impression and (3) the left auricular impression In some cases there is a combination of the impressions produced by the aortic arch and the left bronchus so that there is a single curve above the left auricular curve The impression produced by the pulmonary artery in disease results primarily from the fact that the distended pulmonary stem causes pressure of the left bronchus against the esophagus Such exaggeration of the normal impression produced by the pulmonary artery may occur in congenital heart disease mitral stenosis emphysema and aneurysm of the pulmonary artery

The cardiac sphincter depends on its intrinsic musculature and is functionally independent of the musculature of the diaphragm although the latter may in some cases be an accessory aid in augmenting the sphincteric activity of the cardiac end of the esophagus This independence of the cardiac sphincter is shown by the fact that the sphincteric region of the esophagus may be located above the opening of the diaphragm, and also by the fact that in herniation of the stomach the cardiac sphincter is definitely independent of the diaphragm

The folds of the esophagus are longitudinal and for the most part parallel They vary from two to five in number and may show overlapping of those on the anterior and posterior walls In rare cases they may be undulating and are occasionally decussated The folds may normally be noted through the extreme cardiac end of the esophagus and their demonstration in this region may assume considerable diagnostic importance in the differentiation of a benign from an infiltrating lesion

TECHNIC OF ROENTGEN EXAMINATION

As a precaution before examining the esophagus it is essential that the patient reporting in the morning shall have had no food since the preceding evening The reason is that food particles retained in the esophagus may produce translucent areas within the barium suspension these may be misinterpreted as being the result of intrinsic disease within the esophagus itself Such misinterpretation is particularly likely to occur in patients with cardiospasm Food retained at the extreme cardiac end may distort the contour and produce an irregularity simulating that of actual new growth

In some cases direct fluoroscopic obser

3

The Normal Esophagus

ANATOMIC CONSIDERATIONS

The esophagus is about 25 cm long It extends from the lower end of the pharynx to the cardiac end of the stomach, passing through the superior and posterior mediastinum and piercing the diaphragm at the level of the tenth thoracic vertebra

Liquids on entering the esophagus may reach the lower end in one tenth of a second *Solid or semisolid food is forced down* by peristaltic activity, as originally described by Cannon and Moser[1] in 1898 The circular muscle is constricted from above downward by the peristaltic wave which takes about 5 or 6 seconds to reach the cardia For this purpose of rapid contraction the muscle in the upper part of the esophagus is striated This type of muscle fiber gradually disappears until in the lower portion only smooth muscle is *found There appears to be no pause in the* downward peristaltic movement about 6 seconds being taken for the passage of the wave until it reaches the stomach

The factor of gravity in the swallowing mechanism was described by Schrieber who examined patients during the act of swallowing a bismuth suspension while standing on their heads his purpose being to determine the role of gravity in the act of deglutition He stated that under these circumstances the act of swallowing was insufficient to overcome the factor of gravity and the bismuth suspension could not be forced beyond the proximal area of the esophagus

That the act of swallowing may be successfully accomplished with the patient standing on his head was shown by Thomas[3] The patient was observed fluoroscopically and the rate of progress of the opaque cream through the esophagus into the stomach appeared to be essentially like that taking place with the patient standing on his feet A pictorial presentation of the patient standing on his head and of the roentgenogram which was taken accompany the article The entire esophagus is well outlined *and the opaque material may be noted* making its exit through the cardia and entering the stomach The importance of the factor of gravity however, is quite obvious and in the erect position aids materially in the rapid descent of the swallowed substance through the pars cardia into the stomach

Although the general direction of the esophagus is in a vertical line there are two deviations in its course one is near the *beginning where it deviates to the left* toward the root of the neck and the other is also to the left as it makes its way through the esophageal hiatus

Areas of normal physiologic constriction are present

1 At the junction of the pharynx and esophagus at the level of the cricoid cartilage

2 At the crossing of the aorta

3 In the region where the esophagus passes through the hiatus of the diaphragm The degree of constriction here will depend upon the physiologic state of the diaphragm The narrowing will be less marked if the muscle fibers of the diaphragm are relaxed

At times a fourth area of constriction

suspension into the trachea and bronchi by way of the pharynx In some cases when the obstruction is extreme as thin a mixture of barium as possible is advisable in order to prevent regurgitation

The nature of the mixture and the position of the patient during fluoroscopy and roentgenography are ordinarily essentially identical regardless of the character of the organic lesion of the esophagus which is suspected In special cases such as for example in the roentgen demonstration of esophageal varices further details in the technical management are essential Such variations in technic however will be discussed in connection with the particular problem involved

In the roentgen examination of the esophagus the position of the patient is of considerable importance At the very outset the patient is placed behind the fluoroscopic screen in the erect position For visualization of the esophagus however this position is ordinarily unsatisfactory since the esophagus for the most part is obscured by the shadow of the heart In order therefore to isolate the esophagus and bring it into clear relief the patient is turned obliquely so that his right side is against the fluoroscopic screen a position commonly referred to as the "first oblique" Not only is this position ideal for the study of the esophagus when intrinsic lesions of this region are suspected but it is also important in the study of the stomach as well as will be discussed more fully later As the barium is swallowed each part of the esophagus is closely scrutinized down to the cardiac end As in fluoroscopy in general the screen should be focused as much as possible so as to prevent the escape of unnecessary radiation as well as for increased clearness of detail

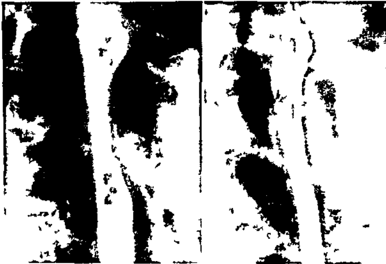


Fig 11 (A *Left*) Air bubbles in the esophagus (B *Right*) Same case as is shown in (A) Note the complete disappearance of the air bubbles at this time

Following observation in the first oblique position the patient should be turned through every angle of obliquity since lesions of the esophagus not clearly demonstrable at one angle may thereby be made visible at another

Films taken after completion of the fluoroscopic study can be taken in both the erect and the prone positions The prone position however is ordinarily preferable the patient being turned through that de

vation of the esophageal area may yield valuable information prior to the administration of any opaque substance for its visualization This is particularly true in some cases of cardiospasm, in which the hugely dilated esophagus may be made visible by the retained fluid within it As a rule, however, the administration of opaque material for the delineation of the esophagus is essential

ever, will pass through the esophagus much more slowly *Prolonged visualization* of the esophagus is thereby aided and films may be taken which will more readily demonstrate any lesion that may be present

The mixture employed at Bellevue Hospital is made up of barium sulfate and acacia, a sufficient amount of the acacia being added to the barium sulfate to thicken it so that it just barely runs off the spoon



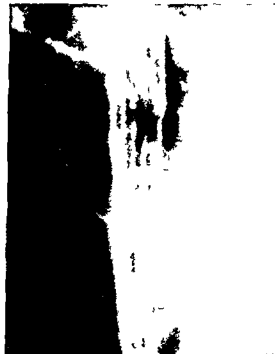


FIG 9 (*Left*) Normal esophagus showing from above downward (1) the aortic impression, (2) the left bronchus impression and (3) the left auricular impression
FIG 10 (*Right*) Normal mucosal folds of the esophagus

In those cases in which the esophagus is examined in routine fashion as part of a study of the stomach (the details of which will be discussed later) no special mixture is employed other than that for the stomach itself When a special study of the esophagus is to be made because a lesion is suspected, a thicker mixture of barium is needed The usual barium water mixture for the study of the stomach passes through the esophagus with considerable rapidity and a detailed prolonged visualization of an organically involved area may be difficult or impossible A thicker mixture how

This should be stirred thoroughly until completely homogeneous Small discrete masses of barium may obscure the picture and give a misleading appearance to the roentgenogram A mixture of this consistency will stick to the esophagus long enough for proper visualization and only a very small quantity is needed As little as one teaspoonful of this mixture will clearly delineate an infiltrating new growth Moreover when the lesion is of a markedly obstructive nature only a small amount of the barium should be administered since, otherwise there may be a reflux of the

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Films taken after completion of the fluoroscopic study can be taken in both the erect and the prone positions The prone position however is ordinarily preferable the patient being turned through that de

gree of obliquity which preliminary fluoro scopic observation has shown to be best suited for the clear demonstration of the lesion

Spot film technic may enhance the value of the radiographic exposure by fixing on the film the exact area of the esophagus under suspicion, as determined by fluoro scopic examination

Figure 9 shows the normal esophagus exhibiting from above down (1) the aortic impression, (2) the left bronchus impres sion and (3) the left auricular impression

Figure 10 shows the presence of the longi tudinal folds of the esophagus

Small globules of swallowed air may be trapped within the esophagus and may simulate polypoid lesions However, the onward passage of these globules may be noted during fluoroscopy and repeated roentgenograms will show the transient character of the alterations

Figure 11A shows small translucent glob ules within the esophagus

Figure 11B shows the disappearance of the globules indicating their transient char acter, the result of swallowed air

A study of the roentgen appearance of the esophagus in normal infants was made at Bellevue Hospital in 1944[6] The study was based on the examination of 32 infants, ranging in age from 3 weeks to 22 months, all of whom were free of gastro intestinal symptoms The observations showed that the esophagus in infants is markedly dilatable and, during the course of the feeding of the barium acacia mixture the lumen was often as wide or even wider than the vertebral column, approximating the di ameter of a normal adult Such variation in the potential distensibility of the esophagus in infancy within the range of the normal must be considered before one commits one self to a diagnosis of cardiospasm More over, regurgitation of the barium from the stomach into the esophagus may give rise to a false conception of abnormal retention, due to a pathologic condition of the cardiac sphincter, a point to be determined only by fluoroscopic observation

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4

Congenital Anomalies of the Esophagus

ANOMALIES OF POSITION

Displacement Displacement of the esophagus may result from congenital anomalies in the position of the right or left subclavian artery

Davis Bayford[1] in 1794 described as dysphagia lusoria the dysphagia produced by the pressure on the esophagus of an abnormally situated right subclavian artery In Bayford's case the right subclavian artery crossed between the esophagus and the trachea, causing constriction of the esophagus with difficulty in swallowing and eventual death from inanition

A reproduction of the anatomic findings in Bayford's case is to be found in Quain's famous atlas of lithographic drawings of the anatomy of the arteries

Among the earliest cases reported of a transposition of the aortic arch were those of Combes and Christopherson[3] and of Herringham[4] In Herringham's case the aorta passed upward to the right of the trachea and crossed to the left behind the esophagus to reach the usual position of the descending aorta on the left side of the vertebrae The patient had complained of dysphagia and of marked dyspnea The findings at autopsy explained the symptoms by demonstrating mechanical compression produced by the abnormally situated aorta which had become dilated in part of its course

A case of congenital anomaly of an aorta with a double arch has been reported by Shaw[5] This is a reproduction of the arrangement usually found in reptiles In his case the two divisions of the aorta united behind the esophagus

In some of the cases of right aortic arch displacement of the esophagus is produced by the pressure of a left subclavian artery originating on the right side and then passing behind the esophagus[6]

Apparently, the first roentgen description of a right sided position of the aortic arch is to be found in the paper by Mohr[7] Two cases are included with the actual roentgenograms showing the aortic arch on the right side Roentgen examination of the esophagus was not made in either case

Illustrations of compression and displacement of the esophagus by a right aortic arch may be found in the contributions of Arkin[8] Renander[9] Loweneck[10] and Korner[11]

Roentgenologically the evidence of displacement of the esophagus by a right aortic arch consists in (1) anterior displacement of the esophagus usually associated with narrowing occurring in the region of the aortic arch and (2) the demonstration of the right sided position of the aortic arch as the causative factor This can best be done by a film taken in the right oblique position

Illustrative Cases An example of a right aortic arch causing anterior displacement of the esophagus and dysphagia is illustrated in the following case

M H aged 40 The patient a woman, complained mainly of a burning substernally and in her throat There was some difficulty in swallowing There was no loss

of weight At times she complained of hoarse ness

Roentgen examination (Fig 12) showed marked anterior displacement of the esophagus by a right aortic arch The course of the aortic arch in relation to the esophagus was well shown (film taken in the first, or right oblique, position) Below the anteriorly displaced portion of the esophagus was a large diverticulum springing from the anterior wall A short distance below this area was another incompletely filled diverticulum



Fig 12 Note the anterior displacement of the esophagus by the right aortic arch Also note the presence of diverticula

The diagnosis of right sided aorta may be aided by contrast roentgenography of the great vessels according to the technic of Robb and Steinberg[1] In this manner the indirect findings of pressure on the esophagus are fortified by the more accurate visualization of the aorta itself This is illustrated by the findings in the examination of one of our patients at Bellevue Hospital

T F aged 50 The patient had entered the hospital complaining of an acute upper respiratory infection Examination was essentially negative except for the teleoroentgenogram (Fig 13A), which showed a huge sacular dilatation of either the ascending or the descending portion of the aorta, and the diagnosis of aneurysm was considered The contrast roentgenogram (Fig 13B) showed that the abnormal appearance was due to a right sided aorta with elongation and tortuosity of the descending portion This was further confirmed by roentgen examination of the esophagus (Fig 13C) showing displacement anteriorly and to the left produced by the anomalous position of the aorta

Snelling and Erb[13] described a case of constriction of the esophagus at the level of the third and fourth dorsal vertebrae produced by a double aortic arch At autopsy it was found that the aorta arose in the usual manner from the left ventricle Three centimeters from its origin the aorta divided into two One branch passed to the left of the trachea and the esophagus and the other to the right of these structures These two divisions united posterior to the esophagus, forming the descending aorta The esophagus and trachea were thus enclosed in a vascular ring which caused the constriction of the esophagus seen in the roentgenographic examination

The manner in which various anomalies of the aortic arch may compress the esophagus and trachea was graphically portrayed by Gross and Ware[14]

ANOMALIES OF FORMATION

Types of Congenital Malformation Ballantyne[15] in his classic work has divided congenital malformations of the esophagus as follows

1 Complete absence of the esophagus its place being taken by a thin muscular band stretching from the pharynx to the cardiac end of the stomach
2 Termination of the esophagus in a simple cul de sac
3 Termination of the esophagus in a cul de sac with the trachea or bronchi
4 Tracheo-esophageal fistula the esophagus being otherwise normal
5 Membranous obstruction of the esophagus



FIG 13 A Right sided aorta—appearance of chest



FIG 13 B Same case as is shown in Figure 13 A Contrast *roentgenogram showing the right sided aorta*

6 Presence of esophageal diverticula
7 Duplicity of the esophagus

When the embryo is about 4 mm long there are two lateral indentations, one on each side of the entodermal pouch These gradually become deeper, finally meeting in the center, thereby forming a septum which divides the pouch into an anterior



Fig 13 C Same case as is shown in Figure 13 A and B Displacement of the esophagus by the right sided aorta

and a posterior tube This septum formation is complete when the embryo is 11 mm in length The respiratory system develops from the anterior tube and the alimentary tract from the posterior tube It is believed that as a result of the incomplete development of the septum a fistulous communication may remain between the trachea and the esophagus

Atresia The most serious congenital anomaly of the esophagus is that produced by atresia This condition may be associated with a fistulous communication with the trachea The comparative rarity of the disease is shown by the fact that only 9 cases of atresia of the esophagus were found in our Bellevue Hospital series of 22,810 autopsies from 1905 through 1935

Atresia of the esophagus with tracheoesophageal fistula in the embryo has been described by Plass [16] Ysander[17] and Gruenwald [18] Ysander's case occurred in an embryo monster 8 mm in length of which he showed a model demonstrating a double esophagus and trachea on each side with a bilateral reduplication of atresia with tracheo esophageal fistula The upper part of the esophagus on each side ended in a

blind pouch The lower portion of the esophagus communicated with the corresponding trachea Both esophagi then entered a common stomach

Habershon[19] described the remarkable case of a cyclopean monster in which the viscera of a double fetus existed in a single peritoneal cavity There was a double esophagus united in a single stomach

Keith [0] described cases of marked narrowing of the beginning of the esophagus similar to a fibrous stenosis of the pylorus The circular muscle was atrophied and was contracted and replaced by loose connective tissue There was no clear evidence of cicatrization He al o described cases of congenital stenosis of the esophagus in which the part that was in the posterior mediastinum was reduced to a fibrous imperforate cord

Atresia of the esophagus may be pre ent without any associated tracheal fistula When a fistula is present the communication may be between the upper segment of the esophagus and the trachea or between the lower segment and the trachea or the trachea may communicate with both upper and lower segments The commonest variety is the one which exhibits a fistula between the terminal portion of the esophagus and the trachea

Roentgen Diagnosis of Atresia of the Esophagus When atresia of the esophagus is suspected examination by the introduction of a barium suspension is contraindicated since it may spill over from the blind pouch into the trachea and lungs The corroboration of the clinical impression of atresia may be accomplished by the introduction of a small nasal catheter and its arrest noted by roentgenographic examination Air may be introduced by means of a syringe and the distended sharply delimited portion of the esophagus may be visualized in this manner A small amount of lipiodol may be introduced through the nasal catheter under fluoroscopic control, and when the roentgen examination has been completed it may be promptly a pirated

When an examination is made the appearance of the abdominal cavity should also be observed and included in the roentgen examination of the chest When a fistula exists between the trachea and the distal end of the esophagus, large amounts of air will be found distending the stomach and

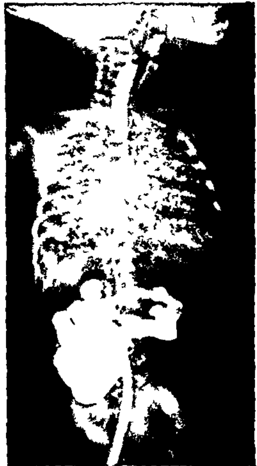


Fig 14 Atresia of the esophagus

intestine having reached the alimentary tract by way of the inspired air If the fistulous communication is limited to the upper blind pouch there will be no air in the alimentary tract Such absence of air however, does not necessarily always preclude the existence of a fistula between the trachea and the distal esophagus An unusually small communication between the trachea and a narrowed lower segment of

the esophagus may prevent the entrance of air [1]

Illustrative Cases Congenital atresia of the esophagus is illustrated by the following case

B P (Fig 14) This child had been born 12 days previously Since birth it had not been able to retain its feedings the food being immediately regurgitated A gastrostomy was done About ½ ounce of sterile water was given through the gastrostomy tube The child became cyanotic started to gag and was in poor condition for the next 5 minutes This was very suggestive of a communication of the lower end of the esophagus with the trachea A second attempt about an hour later produced even more severe attacks of cyanosis with cessation of respiration which was asphyxial in character

The autopsy report stated that ' the esophagus is divided into two portions which are entirely separate and distinct The first is an upper portion, which is a tube with a saccular end This lower end is entirely closed and terminates at the level of the bifurcation of the trachea From the dilated closed end there comes forth a slender strand of fibrous tissue This separate and distinct upper portion of the esophagus does not communicate in any way with the trachea, and its mucosa appears entirely natural The lower portion of the esophagus comprises the inferior two thirds it begins at the bifurcation of the trachea and terminates at the cardiac opening of the stomach The origin comprises the tracheoesophageal fistula and, except for a very light curvature where the esophagus blends with the bifurcation of the trachea the two structures might well be considered as a continuous



Fig 15 A Atresia of the esophagus Note the gas in the alimentary tract

straight tube Thus when a probe is passed through the gastrostomy it passes through the cardiac opening of the stomach up the inferior portion of the esophagus out through the trachea and emerges from the larynx'

From the above description it may readily be seen that there is no possibility that the food taken by mouth will enter the stomach and there i a great possibility that food given by way of the gastrostomy will enter the bronchi and the lungs In fact the lungs present a rather speckled grayish white appearance the



Fig 15 B Atresia of the esophagus

white areas being a few millimeters in size and surrounded by congested lung tissue These white areas appear to represent areas of as pirated barium A careful earch of the other viscera fails to reveal any other congenital anomalies

Roentgen examination of the esophagus following the introduction of lipiodol by way of the mouth showed complete obstruction of the esophagus at the level of the third rib There was moderate dilatation of the esophagus above this point Under fluoroscopic examination lipiodol was injected into the stomach through the gastrostomy tube The stomach was very small and the lipiodol passed over into the coils of small intestine A barium mixture was then introduced into the stomach The barium passed into the esophagus to the level of the fourth dorsal vertebra which portion was about 1 inch below the lowermost portion of the closed off esophagus as shown by the residual lipiodol injected the preceding day At this point the barium passed into the bronchial tree and soon spread into the minute ramifications of the lungs (Fig 14)

The diagnosis was as follows (1) atresia of the esophagus at the level of the third dorsal vertebra (2) esophageal tracheal fistula the fistula occurring between the lower portion of the esophagus and the trachea

The following is an example of atresia of the esophagus with air in the alimentary tract

A K infant 11 days The infant was brought to the hospital because it had been regurgitating its food since birth

Operation revealed an atresia of the proximal esophagus and a tracheo esophageal fistula involving the distal segment of the esophagus The gap between the two ends of the esopha

gus was too great to permit anastomosis The proximal end was therefore brought out to the surface of the skin The distal end of the esophagus was tied off A rubber tube was inserted into the stomach A gastrostomy was done

A small amount of iodochloral (27 per cent iodine in refined peanut oil) was instilled into the esophagus through a rubber catheter, and a *roentgen examination was made Figure 15A* shows the blind termination of the upper segment of the esophagus A small amount of the iodochloral entered the trachea Gas is present throughout the alimentary tract

Operation *revealed that the proximal end* of the esophagus consisted of a blind pouch *which terminated at about the level of the* third dorsal vertebra There was a fistulous *communication between the distal* segment of the esophagus and the right main bronchus

Roentgen examination (Fig 15B) shows the arrest of the lipiodol in the blind pouch formed by the proximal portion of the esophagus A small amount has spilled over into the bronchial tree Gas was noted in the stomach and intestine The preoperative diagnosis was atresia of the esophagus with a fistula between its distal portion and the trachea The diagnosis was confirmed at operation as already noted except that the fistula communicated with the right bronchus



Fig 16 Congenital narrowing of the esophagus

The diagnosis was atresia of the esophagus with an upper blind pouch The presence of gas in the *intestine indicated that there was a* communication between the distal segment of the esophagus and the trachea (tracheo esophageal fistula) These findings were confirmed at operation as noted above

In the next case of atresia of the esophagus *the fistula communicated with the right* main bronchus This premature female infant (E S) *one of twins regurgitated* everything from birth and nurses noted that the gavage tube *was passed with difficulty,* and that the feeding returned immediately by way of the mouth *and nose*

CONGENITAL NARROWING OF ESOPHAGUS

Narrowing of the esophagus other than that due to actual atresia may also be of congenital origin

Roentgenologically, the esophagus in this

condition does not show the blind sac type of termination noted in actual atresia Although the esophagus is narrowed the lumen remains sufficiently wide so that nutrition is not seriously interfered with There is no evidence of an esophageal tracheal fistula In the cases I have seen the narrowing involved the distal portion





Fig 17 (A *Left*) Congenital narrowing of the esophagus (B *Right*) Same case as is shown in (A) Appearance after instrumental dilatation

of the esophagus for approximately a third of its length The contour was entirely smooth

The diagnosis of congenital stenosis of the esophagus other than atresia depends therefore on the following triad

1 Roentgen evidence of a diffuse narrowing of the distal portion of the esophagus

2 Smoothness of outline of this stenosed area

3 A history of difficulty in swallowing since birth

Illustrative Cases Congential narrowing of the esophagus compatible with life and a fair degree of health is illustrated by the following cases

J T, female aged 7 years When this child was 3 days old she began to have projectile vomiting after every feeding The formula was changed repeatedly to no avail At 1 year she weighed only 13 pounds She was hospitalized repeatedly for long periods without any improvement In the past 1½ years she had been having rather severe abdominal pain Esophagoscopy revealed an ulcerated area which bled easily Repeated examinations showed multiple ulcers in and about the cardia with stricture of the distal esophagus This was successfully dilated Further esophagoscopy at a later period showed a widening

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A small amount of iodochloral (27 per cent iodine in refined peanut oil) was instilled into the esophagus through a rubber catheter, and a roentgen examination was made Figure 15A shows the blind termination of the upper segment of the esophagus A small amount of the iodochloral entered the trachea Gas is present throughout the alimentary tract

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Roentgen examination (Fig 15B) shows the arrest of the lipiodol in the blind pouch formed by the proximal portion of the esophagus A small amount has spilled over into the bronchial tree Gas was noted in the stomach and intestine The preoperative diagnosis was atresia of the esophagus with a fistula between its distal portion and the trachea The diagnosis was confirmed at operation, as already noted except that the fistula communicated with the right bronchus



Fig 16 Congenital narrowing of the esophagus

The diagnosis was atresia of the esophagus with an upper blind pouch The presence of gas in the intestine indicated that there was a communication between the distal segment of the esophagus and the trachea (tracheoesophageal fistula) These findings were confirmed at operation, as noted above

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and a pouch formation 22 cm from the teeth with an area of constriction just distal to it Dilatation of the areas of constriction was carried out through the esophagoscope with improvement The roentgen examination (Fig 16) revealed a smooth narrowing of the distal half of the esophagus Because of the onset of symptoms at birth this may justifiably be considered a case of esophageal stricture of congenital origin

Another example of narrowing of the esophagus of congenital origin is illustrated by the following case

R W, male, aged 3 years This was the first admission to the hospital of this 3 year old boy with progressive marasmus and anemia The infant was a full term, 7½ pound baby, delivered by forceps with no apparent birth injury From birth, the infant did not take his formula well and gained weight slowly Development up to 6 months did not appear to be much retarded, as he had his first tooth and could sit up He stood at 11 months but never walked or talked and seemed to have made little progress after the first 6 months At the time of admission, he weighed only 16 pounds 9 ounces vomited frequently and refused solid foods The mother always had to dilute such foods as bananas, mashed potato and pureed vegetables before the baby would take them Six months before admission, the child refused everything but milk and cereal The mother definitely denied that the child had ever swallowed a caustic

On examination he was found to be a veritable bag of bones very irritable with marked pallor

A gastrostomy was done following which the child improved Irrigations of the esophagus were done frequently

Two months after the gastrostomy esophagoscopy showed a tight stricture at the distal end admitting only a number 10 bougie There was no edema or granulation at the site of stricture The stricture was repeatedly dilated through the esophagoscope with success and the patient was discharged considerably improved The child was mentally defective

Roentgen examination of the esophagus prior to the gastrostomy (Fig 17A) revealed almost complete obstruction at the junction of the lower and middle thirds with marked dilatation proximal to this narrowed region A trace of barium was noted passing through the distal portion of the esophagus It was my opinion that the findings could be best explained on the basis of a congenital narrowing and that the almost total constriction at this time might be due to superimposed obstruction caused by retained food

The roentgen examination after the stricture had been dilated (Fig 17B) showed a marked diminution in the degree of constriction although considerable narrowing was still present

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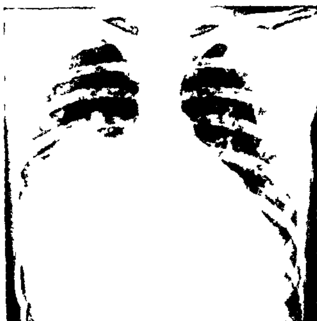


Fig 18 (A *Left*) Marked displacement of the esophagus by a massive left auricle (B *Right*) Massive left auricle (same patient as shown in Figure 18 A)



Fig 19 A Huge aneurysm of the descending aorta

5

Displacement and Compression of the Esophagus by Extra-esophageal Lesions

CARDIAC DISEASE

Marked dilatation of the left auricle causes a displacement in the position of the esophagus posteriorly. As a rule such displacement does not produce sufficient compression to give rise to actual clinical manifestations of dysphagia. In one case, however, the difficulty in swallowing was so marked that a clinical diagnosis of carcinoma of the esophagus was made. Prior to operation, however, fluoroscopic study showed the cause to be marked compression of the esophagus by an extremely enlarged heart. The patient was treated medically for her cardiac condition with prompt alleviation of her dysphagia.

The first studies on the relation of the position of the esophagus in cardiac enlargement were those of Kovacs and Stoerck,[1] Gabert and Rosler and Weiss.[3]

Rigler,[4] in this country, showed the change in position of the esophagus produced by abnormalities of the heart and aorta. Brown and McCarthy[5] made a comprehensive study of alterations from the normal position of the esophagus produced by various conditions such as thoracic deformities, pleuro pulmonary affections, mitral valve lesions, aortic valve lesions, general enlargement of the heart, congenital heart disease, dilatation of the aorta and pericarditis.

In the presence of mitral valvular disease the esophagus is displaced to the right and posteriorly. The explanation for the displacement to the right is that there is a rotation of the heart due to enlargement of the right ventricle. The posterior displacement is a direct result of the pressure exerted by the enlarged left auricle. The enlarged left ventricle may also participate. When both the left auricle and the left ventricle are enlarged, the displacement may affect the entire course of the esophagus in relation to the heart. Pericardial effusion produces only slight displacement of the esophagus. Cardiac enlargement in the presence of lesions of the aortic valve displaces the esophagus to the left and backward.

Schwedel and Gutmann[6] compared the roentgenologic findings with those at autopsy by means of a life size model made of molding clay. They demonstrated the exact relationship of the heart and aorta to the displacement of the esophagus in the roentgenogram.

Kyphoscoliosis may cause considerable change in the position of the upper and middle portions of the esophagus. Other causes of external origin which may produce considerable displacement and compression of the esophagus are aneurysm of the aorta and tumors and abscesses of the lung and mediastinal lymph nodes.

Illustrative Case. The degree to which displacement of the esophagus may occur as the result of cardiac enlargement is illustrated in Figures 18A and 18B.

This patient, in addition to clinical evidence of serious cardiac disease, had complained of difficulty in swallowing for a period of 2 years



FIG 20 A Tumor of the lung—bronchogenic carcinoma



FIG 20 B Same case as is shown in Figure 20 A Compression of the esophagus by the tumor

In this case the posterior displacement of the esophagus (Fig 18A) was produced by a massive left auricle (Fig 18B) The findings were confirmed at autopsy

AORTIC ANEURYSM

Compression and displacement of the esophagus may occur as a result of aneurysm of the aorta The roentgen characteristics



Fig 19 B Same patient as shown in Figure 19 A Marked anterior displacement with compression of the esophagus by an aneurysm of the descending aorta

of this are well illustrated by the following case

Illustrative Case C P male aged 47 years This patient had a history of chancre at the age of 15 He was well until 15 months before his death His main complaint was a severe radiating knifelike pain in the left chest posteriorly and in the lower thoracic spine A nerve block was done to alleviate pain Toward the end the patient complained of difficulty in swallowing

Roentgen examination (Fig 19A) revealed a huge aneurysm of the descending aorta at the level of the seventh to tenth dorsal vertebra There was considerable anterior displacement with compression of the esophagus (Fig 19B)

Autopsy revealed an aneurysm of the descending aorta The aneurysm measured 6 inches transversely, 6 inches in its longitudinal axis and about the same in its anteroposterior extent The mass occupied the major portion of the posterior part of the thorax from the left posterior axillary line to the angle of the ribs on the right The esophagus occupied its normal position above the mass, then curved out forward and to the right over the aneurysm returning to its normal position at the diaphragmatic hiatus There was erosion of the ribs and of the mid dorsal vertebrae

EXTRA ESOPHAGEAL TUMOR

Posterior displacement of the esophagus by a carcinoma of the bronchus is illustrated by the following case

Illustrative Case R C male aged 49 During the 2 weeks before admission he had become increasingly weak short of breath and hoarse and had complained of cough of pain and discomfort in the anterior chest He also had difficulty in swallowing solid but not soft food or fluids Physical examination of the neck disclosed a mass of posterior cervical nodes on the left side just behind the lower end of the sternomastoid They were very hard but not fixed or tender

Autopsy disclosed a bronchogenic carcinoma (oat cell type) of the right main bronchus with metastases to the mediastinal and cervical lymph nodes lungs right perirenal tissues and right kidney, and compression of the trachea bronchi esophagus and great vessels of the mediastinum and neck The esophagus throughout its course was free of any infiltration

Roentgen examination of the chest (Fig 20A) showed evidence of a pulmonary neoplasm Examination of the esophagus (Fig 20B) revealed considerable narrowing near the junction of the lower and the middle third The esophagus was displaced posteriorly in this region and the contour of this narrowed area was essentially smooth The narrowing and posterior displacement of the esophagus were considered to be of extra esophageal origin produced by the pressure of the bronchogenic tumor

Stenosis and irregularity of the contour of the esophagus may be caused by metastatic nodes from a bronchogenic carinoma

Illustrative Case A Z male aged 45 Two months before admission to the hospital the patient developed difficulty in swallowing



Fig 20 A Tumor of the lung—bronchogenic carcinoma



Fig 20 B Same case as is shown in Figure 20 A Compression of the esophagus by the tumor



FIG 21 Constriction of the e ophagus by metastatic nodes from carcinoma of the lung

solid foods but was able to take fluids During the preceding months he also had difficulty in talking and spoke in a husky whisper Also during the past few months he had a chronic cough with sputum that recently had been blood streaked He had night sweats extreme weakness weight loss and some precordial burning pain which was almost constant and which radiated to the left shoulder He had imbibed from 1 to 1½ quarts of whisky a day before the present episode He had lost 20 pounds in the preceding 2 months

Physical examination showed a chronically ill emaciated individual Numerous nodes were palpable in the neck and in particular one firm hard node in the left supraclavicular region not freely movable

Esophago copy showed no evidence of any intrinsic organic disease

Bronchoscopy showed no evidence of tumor masses although the chest service believed that a bronchogenic carcinoma was present on the basis of the roentgen examination of the chest

Autopsy revealed an adenocarcinoma of the right lower lobe of the lung with secondary carcinoma of all lobes of the lung There was secondary carcinoma in the tracheo-bronchial lymph nodes with extension into the adven titia of the esophagus with marked com pression stenosis of the esophagus The au topsy report read as follows ' The esophageal mucosa is smooth throughout There is a con striction of the esophagus 18 cm from its origin The esophagus is dilated slightly for 3 cm above this point of constriction At the point of constriction the lumen of the esopha gus is reduced to 1 cm The constriction is smooth At 15 cm from its origin on its pos terior surface, there is a small polyp which measures 6 cm in diame er It is not ulcer ated Its borders are sharp and clearly defined The remaining portion of the esophageal mucosa is completely smooth

Roentgen examination of the esophagus (Fig 21) showed an area of constriction at about the junction of the upper and the middle thirds There was moderate dilatation of the esophagus above this region Just distal to the smoothly outlined portion the esophagus showed irregularity of contour with varying gradations of constriction This narrowed area gradually faded into the more smoothly out lined portions of the esophagus both proximal and distal The possibility of a new growth of the esophagus was considered As disclosed at autopsy the stenosis and irregularity in the configuration of the esophagus were due to compression by metastatic nodes from the carcinoma of the lung



FIG 22 Marked narrowin agus by an extra-esoph

An extra-esophageal tumor as a cause of a smooth constriction of the esophagus is illustrated by the following case

Illustrative Case E J female aged 29 The patient complained of a lump of 3 weeks duration on the left side of her neck There was a moderate unproductive cough Physical examination revealed a firm nontender mass from about 3 to 4 cm in diameter not attached to the overlying skin In addition there were several small shotty lymph nodes The biopsy report was reticulum-cell lymphosarcoma The patient had x ray treatments with disappearance of the nodes She then developed a lump in the left axilla She lost 10 pounds in weight She had knifelike pains in her chest Two months prior to admission to the hospital she noticed that she could not swallow solid food but could swallow milk and soups She had pain and coughed when she tried to swallow solid foods

Esophagoscopic examination showed a definite narrowing 28 cm from the upper teeth A No 18 French bougie could be passed through the stricture The upper end of the stricture was approximately 10 cm from the stomach The stomach was entered with the bougie following which there was a gush of gas from the stomach The esophagus proximal to the stricture was normal

The essential findings at autopsy as far as they relate to the esophageal condition were

Immediately below the bifurcation of the trachea in the left main bronchus a grayish white hard tumor nodule about 2 0 cm by 1 0 cm projects slightly into the lumen of the bronchus It extends posteriorly and is closely adherent to the esophagus The hilar lymph nodes are enlarged firm and oval and are converted into homogeneous gray white tumor masses as are the mediastinal lymph nodes

At a point at the level of the bifurcation of the trachea the lumen of the esophagus narrows suddenly Nothing larger than the tongs of a regulation blunt forceps passes the level of the stricture The stricture which extends for about 1 cm is the result of a complete collarlike encirclement of the esophagus by the tumor nodule described at the tracheal bifurcation The esophageal mucosa is somewhat injected immediately above the stricture but is otherwise natural

Microscopic examination revealed the presence of a primary bronchogenic carcinoma of the left main bronchus

Roentgenographic examination of the esophagus (Fig 22) showed a marked narrowing at about the level of the bifurcation of the trachea The outline of this narrowed region was very sharply defined and was perfectly smooth There was moderate dilatation of the esophagus above this region The extremely smooth character of the outline suggested that the constricting lesion was probably benign

Dysphagia may result from the pressure of protuberances of the vertebrae (Zahn[7] Pincsohn[8] Mosher[9] and Iglauer[10])

The vertebral pathology occurred at different levels In Zahn s cases as also in the case described by Pincsohn the exostoses were in the thoracic vertebrae In the two cases described by Mosher and the one by Iglauer the pathologic process involved the cervical vertebrae

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Fig 21 Constriction of the esophagus by metastatic nodes from carcinoma of the lung

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Fig 22 Marked narrowing of the esophagus by an extra esophageal tumor

6

Diverticula of the Esophagus

TYPES OF DIVERTICULA
PHARYNGEAL ESOPHAGEAL DIVERTICULA
TRACTION DIVERTICULA
TRACTION PULSION DIVERTICULA
TRANSIENT DIVERTICULA
CONGENITAL DIVERTICULA
EPIPHRENIC DIVERTICULA

TYPES OF DIVERTICULA

Diverticula of the esophagus may be classified under the following types

1 *Pulsion diverticula* These occur mainly on the posterior wall at the pharyngo esophageal region

2 *Traction diverticula* These are found most frequently on the anterior wall of the esophagus at about the level of the bifurcation of the trachea

3 *Traction pulsion diverticula* This type represents a combination of two etiologic factors First the primary underlying traction produced upon the esophagus and secondly the effect of the pressure of food within the esophagus upon this diverticulum

4 *Transient diverticula* These are demonstrable only at times they disappear apparently as a result of changes in the functional behavior of the esophagus

5 *Congenital diverticula* These do not result from the operation of any organic cause such as an adhesion but are the result of a developmental anomaly

6 *Epiphrenic diverticula*

PHARYNGEAL-ESOPHAGEAL DIVERTICULA

The earliest descriptions of this type of diverticulum were those of Ludlow in 1767 and Bell in 1816,[1] Meckel in 1832 and particularly Zenker and von Ziemssen[3] in 1877 Because of the classical description of the condition given by these authors this type of diverticulum is frequently referred to as a Zenker diverticulum

That such diverticula are not of congenital origin is indicated by the fact that they have never been found in the newborn or in early childhood and also because in no case was the muscular layer intact Keith[4] emphasized the fact that there is no embryologic background for the formation of these diverticula There is no branchial cleft for their development However there may be a congenital predisposition to their formation which may be due either to a defect in the arrangement of the inferior constrictor muscle or to a disturbance in function in its two parts

The development of these diverticula on the posterior wall at the junction of the pharynx and esophagus may be understood from the anatomic characteristics of this region Between the transverse and the oblique fibers of the cricopharyngeus muscle is a triangular area which is almost free of muscle structure Such an area is therefore anatomically predisposed to diverticulum formation In addition this is the narrowest area of the pharynx and because of the unyielding character of the anterior wall pieces of food and foreign bodies may readily stick in this region of the posterior wall

Thereafter with every act of swallowing resulting from the contraction of the rest of the pharyngeal musculature the food is pressed against this less resistant region The mucosa may thereby be pushed out

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Anteriorly, the hypopharynx shows a superficial irregularity of contour The hypopharynx becomes narrowed where it joins the neck of the diverticulum Anterior to the diverticulum the esophagus is markedly constricted This constriction however does not appear to be due to the pressure of the diverticulum upon it Such areas of constriction are frequently noted in the absence of a diverticulum This area of constriction is the result of the hyperactivity of the cricopharyngeus muscle, which plays an etiologic role in the ultimate development of the diverticulum emerging as it does above the region of the transverse fibers of this muscle

An operatively controlled example of a Zenker diverticulum is illustrated by the following case

Illustrative Case T S male aged 34 Fourteen months before his admission to the hospital the patient ate a frankfurter and 2 days later regurgitated a piece of it From then on he regurgitated food unchanged in character and free of blood sometimes even 48 hours after eating

During this time he also noted gurgling noises in his neck especially after eating at night The condition gradually became more marked and there was considerable gagging upon attempting to swallow solid food He lost 16 pounds in weight

Physical examination of the neck showed a slight fullness on the left side just above the clavicle and posterior to the sternomastoid On palpation there was crepitation during swallowing accompanied by a gurgling noise

Operation revealed a diverticulum at the junction of the pharynx and the esophagus The sac was freed up to its junction with the lower portion of the pharynx and was sutured through the fundus to the sternomastoid muscle close to the upper attachment

Roentgen examination (Fig 24) showed a typical fairly large diverticulum of the pulsion variety on the posterior wall originating at the pharyngo esophageal junction

Figure 25 is an example of a Zenker diverticulum showing a fluid level and air bubble

TRACTION DIVERTICULA

Traction diverticula in contrast to pulsion diverticula are quite common The first description was that of Rokitansky,[5] who reported one in 1842 Dittrich in 1851 showed the frequency of the affection It was Rokitansky however who in 1861 gave a clear description of the underlying pathology and demonstrated the pull by the



Fig 24 Pulsion diverticulum of the esophagus

cicatrization of a bronchial node upon the esophagus as the important factor in the etiology of such diverticula Zenker described 60 cases verifying Rokitansky's fundamental conception

The origin of the traction diverticulum is to be explained as follows An inflammatory focus involves a localized area of the esophagus As the result of scarring and contraction a pull is exerted upon this portion of the esophagus since the inflammatory lesion is attached to less yielding struc

ward, and gradually may become exaggerated in size until the eventual development of a formed sac, in which food may remain after the act of deglutition has been completed The contents of the sac then increase its size by their weight until it extends downward between the esophagus and



Fig 23 Pulsion diverticulum of the esophagus Note its location at the junction of the hypopharynx and the esophagus

the spine In the filled state the sac may compress the esophagus so that no food may pass through it

Roentgen Diagnosis Roentgenologically this type of diverticulum may be noted high up on the posterior wall at the junction of the pharynx and the esophagus the reason for this location has been shown in the description of the etiologic factors involved The contour is smooth It never has a tent shaped or triangular appearance such as is not uncommonly seen in the traction type of diverticulum The obvious explanation of course, is the etiologic factor in the origin of this condition, the cause being entirely pulsion in nature and never the result of the pull of periesophageal adhesions

It is always single Its size varies greatly It may be so small as to be demonstrable only with difficulty, or it may assume a huge size so that it is noted extending downward to a considerable degree through the superior mediastinum When large, it may produce compression of the anteriorly placed esophagus As a result none of the barium may be noted escaping from the diverticulum into the esophagus or when some of the barium does escape, the esophagus is observed as being narrowed and displaced anteriorly as a result of the compression

In the erect position the diverticulum may be seen as sharply circumscribed with a fluid level capped by air Roentgen visualization of the lesion because of its high position may be readily accomplished in examining the patient in the direct postero anterior view However study of the area is also recommended by changing the position of the patient through every degree of obliquity When a small amount of barium is administered the diverticulum may appear in isolated fashion As more barium is administered some may then be noted escaping into the esophagus unless the diverticulum is huge in size and the compression upon the esophagus considerable

The amount of barium administered however should be carefully gauged under fluoroscopic control since an excessive amount may regurgitate up into the pharynx and so enter the trachea

Figure 23 shows a classic example of a pulsion diverticulum on the posterior wall at the pharyngo esophageal junction The hypopharynx is well visualized The posterior wall is smooth and sharply outlined above the location of the diverticulum

various stages in the developmental process may be traced one being small and tent shaped while another may appear larger and rounded The mucosal folds of the esophagus may pass through without any evidence of radiation towards the base of the diverticulum In other cases however actual radiation of the folds of the esophagus may be noted up to the base of the diverticulum The diverticulum itself is free of folds This feature may be of roentgenologic significance when the diverticulum involves the distal portion of the esophagus In the case of a gastric herniation the mucosal folds of the stomach will be preserved whereas in a large diverticulum which because of location might simulate herniation there will be no evidence of mucosal folds

While in cases of traction diverticula the roentgen examination may ordinarily be carried out in the first oblique position it is nevertheless essential that the patient be turned through every degree of obliquity since a diverticulum not visualized at one angle may be demonstrable at another

Rarely, esophageal diverticula may be associated with the presence of a hiatus hernia of the stomach Because of the rarity of this association however and the comparative frequency of traction diverticula it is my opinion that the relationship is purely coincidental and does not prove that there is necessarily an underlying congenital factor in the development of such diverticula

Roentgen evidence of perforation of a traction diverticulum has been described by Hawes[8] Westergren and Ragnell[9] Sigora[10] Gyorge[11] and Bayer[12]

TRACTION PULSION DIVERTICULA

The third type of diverticulum according to our classification namely the traction pulsion diverticulum has already been described incidentally under traction diverticula As stated there this type of diverticulum represents the culmination of the interplay of two factors (1) the pull upon the esophagus produced in the process of cicatricial contraction of an inflammatory node, and (2) the superadded factor of pulsion produced by intrinsic pressure within the esophagus Such a diverticulum therefore represents only an end result of the etiologic process underlying the development of traction diverticula in general The two forces of traction and pulsion in such cases go hand in hand The same factors in roentgen diagnosis apply here as were described for traction diverticula

TRANSIENT DIVERTICULA

Barsony and Polgar[13] considered many of the cases of multiple diverticula of a transient nature as functional and asymptomatic and due to disturbances of innervation in the wall of the esophagus As a result the area of involvement does not contract Because of intra esophageal pressure, such localized areas of involvement became distended and diverticular like in appearance

Evidence in support of a functional factor in the production of this phenomenon is furnished by Knight[14] He used cats because the distribution of striped and unstriped muscle in their esophagi closely simulates that of man On stimulation of the vagi or on removal of the stellate ganglia he produced an appearance of the esophagus similar to that of the globular segmentations in man This is illustrated by a roentgenogram of the esophagus of the cat *exhibiting these changes after the* bilateral removal of the stellate ganglia

Simon[15] suggested that the incomplete development of the musculature of the esophagus or congenital malformations of its tissues might give rise to localized areas of diminished resistance to intra esophageal pressure leading to the picture of transient diverticulosis of the esophagus Simon quoted Ribbert as favoring developmental anomalies as a cause of diverticula of the esophagus and also the work of Havlicek,[16] whose investigations in the development of reptiles and birds have given support to

tures, such as the trachea or bronchi The most common source of inflammation is a bronchial or tracheal lymph node, particularly at the level of the bifurcation It is because of the increased localization of lymph nodes in this region that there is a special predilection of this portion of the esophagus for the formation of the traction type of diverticula

The node may only become adherent to the outer layer of the muscular wall or it



Fig 25 Pulsion diverticulum with fluid level and air bubble

may actually invade this layer With contraction the muscular layer is pulled out and the mucosa follows In other cases the invasion may reach the mucosa Both layers, the mucosa and the muscle, are then pulled outward In other cases the node becomes adherent to the mucosa through an opening in the muscular wall as the result of a periadenitis with abscess formation In the early period of such infection the abscess may actually break through into the esophagus After evacuation of the pus the perforation may then be closed off With later contraction of the cicatricial tissue the mucosa may be pulled out through this gap in the muscular wall This also helps to explain the presence of scar formation in the depth of the mucosa of some diverticula

Roentgen Diagnosis The earliest attempt to diagnose a diverticulum roentgenologically was made by Reitzenstein[6] in 1898 In a patient of his after the administration of a bismuth suspension, the diverticulum was observed on the fluorescent screen, following which roentgenograms were taken

Diverticula of the traction variety are limited entirely to the esophagus itself and have never been found in the pharyngoesophageal region Because of the preponderance of nodes on the anterior wall of the esophagus at about the level of the bifurcation of the trachea, such diverticula are to be found most commonly in this region By the time that roentgen examination is made these diverticula usually appear well rounded and perfectly smooth in outline In size, they may show considerable variation, although they usually do not achieve the size sometimes noted in a Zenker diverticulum In some cases the diverticulum may be tent shaped This apparently represents an early stage in the process of formation before it has become rounded as the result of pressure exerted upon it from within the esophagus itself In some cases, a calcified node may be seen in close apposition to the diverticulum, this presumably represents the etiologic factor involved in its production I have never noted peristaltic activity in a diverticulum of this character presumably partly because of the attenuation of the muscular structure and partly because of the destruction of the layers in the wall of the diverticulum as a result of the inflammatory cicatrizing process

As a rule these diverticula are single They are rarely multiple In 1926 I described multiple diverticula of the esophagus which had been discovered on routine roentgen examination[7]

In some cases of multiple diverticula the

Here, too, Fleischner considered the condition as being due to the presence of multiple adhesions involving the esophageal wall In support of this view Fleischner stated that these wavelike and segmented areas recur in the same location even though they may show variation as to form and size These latter variations may be explained on the basis of differences of intra esophageal pressure associated with peristaltic activity Adhesions however the wall produced by the adherent inflammatory process These alterations are qualitatively different from the encircling types of undulations of the esophagus In this latter phenomenon, there is certainly no roentgenologic evidence of abnormal fixation of the esophagus in any one region There is no permanent narrowing of any segment or of any localized areas of dilatation of a permanent character, nor is the configuration at all similar to the usually



Fig 27 (A *Left*) Diverticulum of the esophagus showing failure of visualization in the right oblique position (B *Right*) Diverticulum of the esophagus visible with the patient in the left oblique position

fixed at various regions throughout the esophagus may explain their constant recurrence in the same location However similar phenomena may be noted when the esophagus is dilated so that in some cases at least the appearance must be considered as being functional Also the circular nature of some of the segments of the esophagus seen in this phenomenon cannot be readily explained on the basis of adhesions and suggests that the etiologic factor may be functional

In mediastinitis the contour of the esophagus may be altered with the production of irregular projections due to the pull upon pointed narrow projections of a highly irregular character seen in association with mediastinitis which remain practically constant and are hardly affected from moment to moment by the behavior of the esophagus Instead of a comparatively small localized segment of the esophagus involved in mediastinitis, the entire esophagus at times may partake of the changes noted in these recurrent usually smoothly outlined undulations

If these changes were produced by periesophageal adhesions one would have to assume first that the process involved practically the entire length of the esophagus

the theory of the congenital origin of these diverticula

Fleischner[17] made out a strong case for traction as the etiologic factor in transient diverticula of the esophagus, rejecting the conception of Barsony that they are functional in nature

Some of his reasons were as follows

1 Such transient diverticula are found as a rule in those areas in which typical traction diverticula are most apt to develop namely, the anterior and lateral wall of the esophagus at about the level of the bifurcation of the trachea

2 In some cases such transient diverticula are found in association with typical traction diverticula

3 Calcified foci are found in the peritracheal lymph nodes near the base of these transient diverticula

4 Such transient diverticula may be found in association with paresis of the recurrent laryngeal and phrenic nerves and in cases of bronchial asthma conditions which may be associated with a mediastinitis

5 An important point in favor of an anatomic basis for these transient diverticula is the tendency for them to recur in the same location due allowance being made for variations in the degree of filling with barium

6 His most important evidence is his confirmation by anatomic findings at the site of these transient diverticula in five cases which were studied at autopsy

In Fleischner's opinion therefore, practically all diverticula including those of a transient character, are secondary to traction except for a few cases of pulsion diverticula based on congenital predisposition



FIG 26 Diverticulum of the esophagus

The question of their functional or organic origin in every case, however, has not been clearly established

An interesting feature in the appearance of the esophagus is that of marked undulation with a tendency to segmentation involving various portions of the esophagus and at times occurring throughout its entire length The appearance may simulate that of small peristaltic waves

In those cases in which there is repetition of the identical pattern one must assume that localized areas of diminished resistance exist at these particular levels perhaps on the basis of deficiencies of muscular structure

The localized transient alterations in the contour are not always of a globular character They may appear serrated or there may be a combination of serrations and wavelike ripples At times the esophagus may assume a corkscrew appearance In addition to vagaries in the developmental character of the muscular structure irritability of the esophagus of functional origin may be an important accompaniment in the exaggeration of these peculiar phenomena

CONGENITAL DIVERTICULA

That some diverticula of the esophagus may be of congenital origin is suggested by the embryologic development of the esophagus originating from the fusion of two segments an upper portion arising from the retropharyngeal segment of the foregut, and a lower portion arising from the pregastric segment of the foregut A primary defect at the point of union of these two segments may be the site of the development of a diverticulum Diverticula on the posterior wall may originate from cysts of the esophagus which are present during embryonal life These may persist into adult life and may acquire an opening into the esophagus with the formation of diverticula Also, as



Fig 30 (A *Left*) Diverticulum with a conical apex Note the irregular serrations of the esophagus (B *Right*) Same patient as is shown in Figure 30 A Note the disappearance of serrations with relaxation of the esophagus

in some cases and that the adhesions completely encompassed the wall. Otherwise, only one part of the wall would be expected to partake in the changes of contour. Since the rounded areas are of globular character *and frequently involve the entire contour*, only adhesions of a completely encircling character running throughout the length of the portion of the esophagus involved should be expected to produce an appearance of this kind.

It is difficult to believe that such a widely disseminated pathologic process would continually escape detection in the examination of the mediastinum in all these cases.

One of the arguments in favor of adhesions as a cause of this phenomenon is the occasional association of a true diverticulum of permanent character, yet we commonly see these changes in the absence of actual traction diverticula, and they may be found in individuals *who are entirely* free of any associated disorder of the esophagus. They may also be present in individuals suffering from other associated organic *lesions* of the esophagus, such as carcinoma. On the whole, the evidence would seem to favor a functional basis for this phenomenon associated with localized areas of lessened resistance throughout the esophagus. Mere irritability of the esophagus alone would hardly explain the constant reduplication of the findings in some individuals at the identical areas during prolonged fluoroscopic observation or in successive roentgen exposures. Also when the examination is repeated at a later date the localized areas of globular configuration may again show recurrence in identical areas.





Fig 28 (*Left*) Traction diverticulum of the esophagus
Fig 29 (*Right*) Traction diverticulum. Note the relation to it of the calcified node

demonstrate the presence of a diverticulum of the esophagus is illustrated by Figure 27A and B In Figure 27A the patient is in the first, or right oblique, position There is no clear evidence of an esophageal diverticulum Examination in the second or left oblique position (Fig 27B) shows definitely a diverticulum originating from the anterior aspect of the midportion of the esophagus and directed downward Note also that there has been regurgitation of a small amount of barium into the trachea Clinically the patient gave a history of coughing spells initiated by the swallowing of liquids

Figure 28 shows a smoothly outlined rounded diverticulum of the anterior wall of the esophagus The mucosal folds of the esophagus run longitudinally and do not radiate into the diverticulum At times however the mucosal folds of the esophagus may be noted radiating into the region of the diverticulum

Figure 29 illustrates a possible etiologic relation between a diverticulum on the anterior wall of the esophagus and the presence of a calcified lymph node in close relation to it Inflammatory pathology within the node may have been the exciting factor in the production of traction upon the esophagus with the eventual development of a diverticulum

G H, aged [illegible]0 The patient had complained of distress occurring from 10 minutes to 1 hour after meals Roentgen examination showed an ulcer of the first portion of the duodenum At one time the patient complained of difficulty in swallowing food

Roentgen examination of the esophagus (Fig 29) revealed a cone shaped diverticulum of the anterior wall at about its midportion At the apex of the triangle was a calcified node which by traction upon the esophageal wall may have been the cause of the diverticulum The mucosal folds within the esophagus were intact

The next case illustrates not only the presence of a definite diverticulum of the esopha

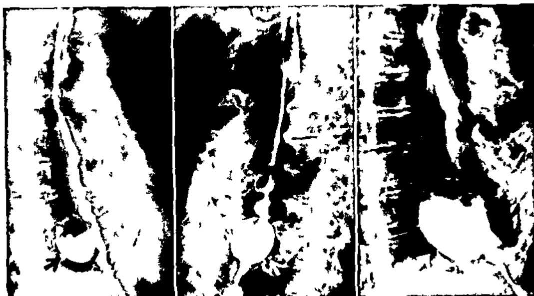


Fig 32 (A *Left*) Diverticulum on the posterior wall of the esophagus There are two small vaguely outlined saccular areas proximal to this (B *Center*) Same patient as is shown in (A) Appearance in left oblique position (C *Right*) Same patient as is shown in (A) and (B) Examination 5 years later Note the marked increase in the size of the diverticulum

the result of imperfect separation of trachea and esophagus during embryonal life, esophageal pouches may develop

Roentgenologically, a diverticulum may



FIG 31 Multiple diverticula of the esophagus Note also the wavelike configuration of the proximal portion of the esophagus

be suspected of being of congenital origin if it occurs elsewhere than on the anterior wall of the esophagus and is perfectly rounded in appearance While the positive identification of a traction type of diverticulum is a much simpler task because of its site of predilection and its occasional tent shaped appearance, any diagnosis as to the possible congenital origin of an esophageal diverticulum on the basis of the roentgen findings must remain entirely in the field of conjecture

EPIPHRENIC DIVERTICULA

A rare type of location for an esophageal diverticulum is illustrated by the epiphrenic variety These are of smooth outline and rounded contour and in many respects have the characteristics of a pulsion diverticulum When associated with cardiospasm, the etiologic factors underlying the development of such a diverticulum may be twofold (1) a weakness may be present in the wall of the esophagus above the cardiac end, and (2) exaggerated intra esophageal pressure as a result of the cardiospasm may cause a bulging of the wall of the esophagus at the site of this area of diminished resistance Except for its rare location the roentgen features of this type of diverticulum are essentially like those produced by pulsion elsewhere

Illustrative Cases The following are examples of various types of diverticula of the esophagus

T C, male aged 71 The patient gave a history of difficulty in swallowing for years which had become more marked in the preceding 2 months There had been continuous loss of weight in the preceding 1½ years

Roentgen examination (Fig 26) revealed a large diverticulum with a broad base springing from the anterior wall of the esophagus There was no evidence of infiltration new growth, or obstruction to the flow of barium at the time of the radiographic study

Following esophagoscopy he developed complete obstruction being unable to swallow even water A gastrostomy was done

The autopsy report read as follows 'On opening the esophagus there is found about 2 cm below the region of the bifurcation a traction diverticulum in the anterior portion of the esophagus This diverticulum measures about 1 5 cm in diameter and about the same in depth It is about 10 cm above the cardiac opening of the stomach'

The importance of examining the patient through various angles of obliquity to

demonstrate the presence of a diverticulum *of the esophagus is illustrated by Figure* 27A and B In Figure 27A the patient is in the first or right oblique position There *is no clear evidence* of an esophageal diverticulum Examination in the second or left oblique position (Fig 27B) shows *definitely a diverticulum originating from* the anterior aspect of the midportion of the esophagus and directed downward Note

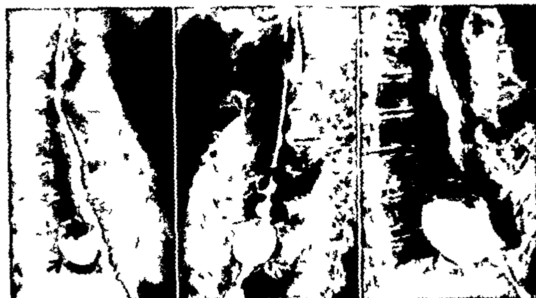


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Figure 29 illustrates a possible etiologic relation *between a diverticulum* on the anterior wall of the esophagus and the presence of a calcified lymph node in close relation to it Inflammatory pathology within the node may have been the exciting factor in the production of traction upon the esophagus with the eventual development of a diverticulum

G H aged 50 The patient had complained of distress occurring from 10 minutes to 1 hour after meals Roentgen examination showed an ulcer of the first portion of the *duodenum* At one *time the patient com*plained of difficulty in swallowing food

Roentgen examination of the esophagus (Fig 29) revealed a cone shaped diverticulum of the anterior wall at about its midportion At the apex of the triangle was a calcified node which by traction upon the esophageal wall may have been the cause of the diverticulum The mucosal folds within the esophagus were intact

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The importance of examining the patient through various angles of obliquity to

the first portion of the duodenum which explained his clinical manifestations The findings in the examination of the esophagus were apparently incidental The esophagus was radiographed on a number of occasions over a period of almost 5 years Figure 32A shows the appearance in the first or right oblique erect position There is a large diverticulum rounded saccular area Because this was not noted in Figure 32A and at other examinations it was believed that it probably represented a transient ampullary dilatation of the cardiac end

Roentgenographic examination 5 years later (Fig 32C) showed a marked increase





Fig 34 (A *Left*) Multiple sacculations of the esophagus (B *Right*) Same patient as is shown in (A) Examination 12 days later Note the tendency to reduplication of the appearance

on the posterior wall of the esophagus a short distance above the diaphragm The diverticulum contains an air bubble occupying the upper half Proximal to this large diverticulum are two small vaguely outlined saccular areas In the second or left oblique position (Fig 32B) the large diverticulum is seen full face Two smaller diverticula are noted proximal to it At the extreme distal end of the esophagus at this time is a small in the size of the diverticulum evidently the result of pulsion

An unusual example of multiple diverticula of the esophagus is illustrated by the following case

A S female aged 40 Four months before her admission to the hospital she had had an obstructive sensation in her esophagus at the

gus but other interesting phenomena as well Roentgen examination revealed the fact that the diverticulum, occupying a characteristic location was sharply pointed at its apex This apparently represents an early stage in the developmental history of an esophageal diverticulum Sharply pointed at first as the result of traction, the contour may eventually become rounded as the result of associated secondary pulsion in addition to the primary traction Another interesting feature of this

In the next case there are three diverticula originating from the anterior wall of the distal portion of the esophagus (Fig 31)

The patient (E G) had no symptoms referable to the esophagus The clinical diagnosis was duodenal ulcer In addition to the diverticula note also the undulations of the proximal portion of the esophagus

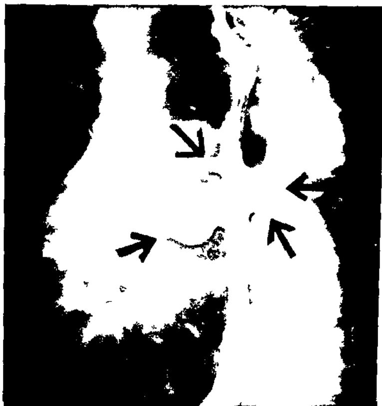


Fig 33 Multiple diverticula of the esophagus

case is the presence of numerous small projections throughout most of the esophagus (Fig 30A) Some of these are pointed others are more rounded in contour That these are of a transient character is demonstrated by their absence at a time when there is complete relaxation of the esophagus although the large diverticulum is still present (Fig 30B)

The cause of these transient irregularities of the esophagus is probably functional irritability This may be fundamentally similar to the phenomena of irritability present elsewhere in the alimentary tract as, for example in the stomach and colon

Rarely a diverticulum may originate from the lower end of the esophagus just above the diaphragm and on the posterior wall In the following case not only does the diverticulum occupy this unusual position but it is associated with two smaller diverticula just proximal to it

G M male aged 67 The patient gave a 10 year history of recurrent attacks of epigastric pain relieved by an ulcer regimen The pain frequently woke him from sleep There were no symptoms referable to the esophagus

Roentgen examination showed an ulcer of

under identical conditions of tone and peristalsis

The superficial undulations in the contour of the esophagus which behave like ripples their pattern changing momentarily, may be an entirely different phenomenon from this type of diverticulosis These superficial ripples may be the expression of irritability or of a disordered form of peristalsis

A peculiar type of anomalous behavior of the esophagus may be noted in figure 35 The distal portion is twisted in corkscrew fashion

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level of the sternum She also complained of occasional epigastric distress She had had intermittent regurgitation during meals for 1 year There was no dysphagia, anorexia or weight loss

Physical examination was essentially negative, except for absent ankle jerks She was under treatment for syphilis

Because of the esophageal symptoms, esophagoscopy was done The instrument was passed without difficulty The mucosa of the esophagus was normal throughout About 22 cm from the teeth (incisura) a large secondary patent opening was found leading off from the right anterolateral wall of the esophagus The opening appeared to be pencil size No pathology of the mucosa was present



Fig 35 Corkscrew esophagus

The diagnosis was 'diverticulum of the esophagus"

Roentgen examination (Fig 33) revealed a rather large diverticulum on the anterior wall of the esophagus at about the level of the bifurcation of the trachea Proximal to this area was a much smaller diverticulum also springing from the anterior wall On the posterior wall of the esophagus at about the same level was a saccular area with a broad base and near its upper margin a small square shaped projection

Apparently, all the esophagoscopist was able to see was the opening leading to the larger diverticulum with its somewhat narrowed neck If this structure is to be explained on the basis of traction, one must assume that there were multiple areas of adhesions, involving both the anterior and the posterior wall of the esophagus at approximately the same level Note that on esophagoscopy there was not the slightest evidence of intraluminal organic disease and that the mucosa was entirely normal throughout

Multiple diverticulosis involving almost the entire esophagus is illustrated by the next case

S Z aged 64 The patient was suspected clinically of having a carcinoma of the tail of the pancreas There was no history of dysphagia The findings on roentgen examination of the esophagus were coincidental At the first examination (Fig 34A), the esophagus showed numerous saccular areas throughout its distal half They involved the anterior and posterior walls and were apparently circumferential On re examination 12 days later this phenomenon was again noted (Fig 34B) Note in particular how closely the appearance in Figure 34B mimics that in Figure 34A We are therefore dealing with a tendency to reduplication in the formation of these diverticula This behavior was strongly suggestive of localized factors at the areas where these phenomena recurred It was difficult to understand how adhesions could produce these findings unless we presupposed that they involved almost the entire length of the esophagus and completely surrounded it thereby exerting almost simultaneous traction on the wall in every direction

Mere irritability of the esophagus does not offer a satisfactory explanation of the tendency to reduplication of the identical pattern Perhaps the explanation that best accounts for this behavior is the one that assumes that there are areas of diminished resistance in the structural integrity of the wall of the esophagus perhaps of congenital origin These concentric bulgings would then tend to recur time and time again in these regions of diminished resistance

scarring of a thickened sphincter hypertrophic oblique muscle of the stomach kinking of the terminal esophagus or of compression of the esophagus by spasm of the diaphragm In 10 cases the vagus nerve was normal But in all cases he found a marked loss or complete absence of ganglion cells from the myenteric plexus The disorder is therefore primarily a disease of the myenteric plexus resulting in failure of relaxation of the esophagus during the normal act of swallowing

One of the less common functional disturbances of the esophagus giving rise to difficulty in deglutition is that of paralytic origin Thus involvement of the recurrent laryngeal nerve may seriously disturb the swallowing reflex Barium may remain at the valleculae in the sinus pyriformis and at both sides of the thyroid cartilage

In some cases of paralysis of the recurrent laryngeal nerve swallowed barium may enter the trachea In some cases of carcinoma of the esophagus associated with the passage of barium into the trachea this may be due to the pressure of a growth upon the recurrent laryngeal nerve Paralysis of the vagus nerve may also give rise to difficulty in swallowing

Von Rosenheim and Holzknecht many years ago described a type of dysphagia due to an intrinsic atony of the esophagus not associated with actual obstruction with barium sticking to the entire length of the mucosa





Fig 36 (*Left*) Cardiospasm with marked dilatation of the esophagus
Fig 37 (*Right*) Cardiospasm with tortuous esophagus

ROENTGEN CHARACTERISTICS

Roentgenologically in cardiospasm the esophagus shows various degrees of dilatation It may become enormous in size, and in routine examination of the chest the appearance may simulate that of a heart that has become tremendously enlarged In foldings of the esophageal contour may be noted Fluoroscopically the esophagus may exhibit exaggerated peristaltic activity in an effort to overcome the failure of relaxation of the cardia

7

Cardiospasm

ROENTGEN CHARACTERISTICS

Among the earliest reports of cases of dilatation of the esophagus were those by Purton[1] Hannay and Rokitansky

That cardiospasm was due to a failure of relaxation of the cardia rather than to an actual spasm in this region was emphasized by Einhorn[3] and Rolleston[4] Rolleston's case is of value in that the findings were checked by autopsy A boy, 8 years old, after an attack of whooping cough, developed intractable vomiting from which he died The autopsy revealed a markedly dilated esophagus which when distended had a circumference measuring 3½ inches At the postmortem examination fluid food was still present in the esophagus The esophagus was 9½ inches long and ran through the thorax in a tortuous manner The muscular coat was hypertrophied The mucosa was normal The lower end of the esophagus was spindle shaped A finger could be passed from the stomach through the esophageal opening No evidence of stricture was present either grossly or on microscopic study The condition was therefore one of primary dilatation Because of the muscular hypertrophy Rolleston believed that a functional obstruction might have been present at the cardiac end during life, due either to inhibition of the contraction of the longitudinal muscle fibers or to spasm of the circular muscle fibers Rolleston furthermore brought up the following interesting point Assuming that the obstruction was spasmodic there should be evidence of hypertrophy of the muscular fibers forming the cardiac sphincter However, no such evidence was present in this case He suggested a failure in the co-ordinating mechanism of the act of swallowing during which the cardiac sphincter is ordinarily relaxed, as explaining the dilatation and hypertrophy of the esophagus Paralysis of the longitudinal muscle fibers of the esophagus would lead to dilatation and by interfering with the relaxation of the cardiac sphincter would eventually lead to hypertrophy of the circular muscular fibers

This condition of failure of relaxation of the cardia has been described by Hurst[5] as achalasia According to him the conception of achalasia of the esophagus' was first broached in the medical literature by Thomas Willis in 1672

In ten specimens of achalasia of the esophagus Rake (quoted by Hurst) found degenerative changes involving the ganglion cells of Auerbach's plexus Achalasia of the esophagus thus appears to be due to organic disease of Auerbach's plexus Another point in favor of the conception of achalasia as opposed to cardiospasm is the fact that the cardiac sphincter has never been found hypertrophied at autopsy Also, in those cases in which operation has been performed, the sphincter within the abdomen is found to be either of normal thickness or actually atrophied even though the condition has been of long standing

This work was confirmed by Lendrum,[6] who made a detailed gross and microscopic study of 13 cases of cardiospasm In all cases he found that the dilated portion of the esophagus was separated from the stomach by a segment which showed no dilatation a region which he called the neck,' varying in length from 1 5 to 4 5 cm There was no evidence of organic narrowing or

scarring of a thickened sphincter hyper trophic oblique muscle of the stomach kinking of the terminal esophagus or of compression of the esophagus by spasm of the diaphragm In 10 cases the vagus nerve was normal But in all cases he found a marked loss or complete absence of ganglion cells from the myenteric plexus The disorder is therefore primarily a dis

passage of barium into the trachea, this may be due to the pressure of a growth upon the recurrent laryngeal nerve Paraly sis of the vagus nerve may also give rise to difficulty in swallowing

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Roentgenologically in cardiospasm the esophagus shows various degrees of dilata tion It may become enormous in size, and in routine examination of the chest the appearance may simulate that of a heart that has become tremendously enlarged In foldings of the esophageal contour may be noted Fluoroscopically the esophagus may exhibit exaggerated peristaltic activity in an effort to overcome the failure of re laxation of the cardia

Differential Diagnosis of the Esophagus in Cardiospasm The contour is smooth and tapering, ending in the cone shaped extremity at the cardiac end This sharpness of outline aids in differentiating the condition from possible malignant infiltration

Obviously, one must attempt to make certain that the esophagus is empty at the time of examination since food particles retained at the cardiac end may produce translucent defects suggesting organic disease

Enormous dilatation of the esophagus speaks invariably for a benign cause When the obstructive lesion is malignant the esophagus, though it exhibits dilatation above the area of involvement as a rule never does so to a very marked degree This is due to two factors In the first place a patient with a malignancy of the esophagus dies long before there has been much opportunity for unusual dilatation, whereas in cardiospasm the obstruction may continue for many years In the second place, the obstruction produced by a new growth is never as marked as in cardiospasm There is usually a sufficient canalization so that fluids may pass through even though solid foods cannot Moreover the sloughing which a malignant tumor frequently undergoes, increases the canalization and consequently diminishes the degree of obstruction, so that unusual dilatation is not quite so apt to occur

Another aid in differential diagnosis is that while in cardiospasm it is the cardiac end which becomes narrowed malignant invasion usually affects other regions of the esophagus and, less frequently, the extreme distal portion Even when this region is involved the process as a rule is not limited to this area but is more diffuse, with evidence of invasion both proximally and distally in the cardiac region of the stomach Destruction of the mucosa in "relief pictures may aid considerably in the differentiation of a malignant infiltrative process from the benign lesion of cardiospasm



FIG 38 (A *Left*) Cardiospasm in a child of 10 (B, *Right*) Appearance of the child 2 weeks after admission to the hospital

Illustrative Cases The following is a case of cardiospasm with a characteristic roentgen appearance and esophagoscopic corroboration

J S aged 30 The esophago cope was passed without difficulty down to its lower end The mucous membrane appeared normal There was no evidence of foreign body or neoplasm Three days later the esophagoscope was passed into the stomach without difficulty No abnormality of the mucosa was noted The cardiospasm was dilated by bougies passed through the constricted area under esophagoscopic control

Roentgen examination (Fig 36) revealed marked dilatation of the esophagus with a fluid level near its upper portion The cardiac end tapered as it entered the stomach and was smooth in outline

From the position of the right border of the dilated esophagus one could readily see how it might simulate when not outlined by barium the right border of an abnormally enlarged heart

The absence of any organic disease associated with the obstruction was demonstrated by esophagoscopic observations

Another excellent example of a dilated esophagus due to cardiospasm is illustrated in Figure 37 Note the infoldings of the esophagus which are not uncommonly associated with marked increase in size Note the smoothly tapering cardiac end of the esophagus with a fine stream of barium making its exit

Cardiospasm while commonly a disease of adult life may in rare instances occur in the very young This is illustrated by the following case

M C female aged 10 Four months before her admission to the hospital this child had contracted pertussis She complained of cough and vomiting The cough ceased a few days before admission The vomiting persisted however and occurred about once or twice a day at times immediately after the ingestion of a meal She did not complain of pain

Roentgen examination indicated the presence of marked "cardiospasm" with dilatation of the esophagus Very little of the barium was noted entering the stomach Within 15 minutes after a hypodermic injection of 1/150 grain of atropine the esophagus was practically entirely empty of barium

Three days later examination of the esophagus 6 hours after the administration of barium (Fig 38A) showed retention in the esophagus of half the barium Thirty minutes after the administration of a hypodermic of 1/150 grain of atropine, the esophagus was again empty of barium

The esophagoscope was passed to just above the hiatus which showed considerable thickening of the mucous membrane At the hiatus there was considerable angulation anteriorly and to the left with spasm which made it difficult to pass the esophagoscope into the stomach

Figure 38B shows the appearance of the child at the time of admission to the hospital

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8

Benign Tumors of the Esophagus

ROENTGEN DIAGNOSIS

Early descriptions of the pathologic appearance of benign tumors of the esophagus have been published by Meckel, Zenker and von Ziemssen and Cruveilhier

The rarity of such tumors is shown in the Bellevue Hospital records during the period 1905 through 1935, which disclosed 16 cases of benign tumor of the esophagus Of these 9 were found at autopsy out of a series of 22 810 autopsies conducted during this period These tumors were classified as follows papillomas, 4 fibromas, 3 myomas 2 lymphangiomas 2 polyp 1 leiomyoma 1 and angioma 1 Two of the tumors found at autopsy were not classified

Patterson[1] *stated that in a review of the* literature from 1712 to 1932 she found 61 cases of benign tumors of the esophagus, to which she added a case of a myxofibroma Only 42 were reported in any detail and even in some of these cases description was incomplete (The article contains no bibliography of references to these cases) Although the lesion is quite rare there has been a considerable increase in the number of cases recorded since 1933 and in the *report by Adams and Hoover in 1945 35* additional cases were recorded in the literature (including 3 of their own) thus making a total of 97 cases To these may be added the 16 cases referred to above found at Bellevue Hospital

ROENTGEN DIAGNOSIS

The essential findings in the demonstration of a benign tumor of the esophagus are the following

1 The tumor usually produces a rounded centrally located translucent area corresponding to the intraluminal position of the growth This is due to the anatomic characteristics of benign tumors generally which hold for tumors of the esophagus as well *This rounded translucent appearance* produced by a benign tumor is shown in the cases reported by Pape and Spitznagel[3] Beutel,[4] and Palugyay Exceptionally, the tumor may be of such nature that the roentgen appearance may simulate that of a proliferating malignant tumor This occurred in Zehbe's[6] case, in which roentgen examination of the esophagus disclosed a thin layer of barium passing along the lateral walls and encompassing several dense areas *of different form and size At* autopsy, however, a large polyp was found, the size of a cucumber which hung by a pedicle from its anterior wall and filled almost the entire esophagus Histologic examination showed it to be a fibroma

The diagnosis is further complicated by the fact that sarcomas of various types may in some cases appear as rounded, well circumscribed intraluminal tumors, sometimes pedunculated Thus the sarcomas re*ported by Ogle*[7] *Albrecht,*[8] *Stephan,*[9] Wolfenberger[10] von Hacker[11] and Dvorak[12] were pedunculated or polypoid

In Sommers case,[13] roentgen examination showed a translucent area within the lumen of the esophagus, outlined by barium Autopsy showed a sausage shaped tumor 14 cm long within the lumen of the esophagus fixed to the wall by a short pedicle Histologic study, however, showed the tumor to be a carcino sarcoma

Similarly, Krieglstein[14] described the roentgen findings of an intraluminal ellipti

cal area within the esophagus the size of a goose egg outlined by barium which at autopsy proved to be due to a fibroma with sarcomatous changes

1 While exceptionally therefore the differential diagnosis between a benign tumor and a malignant lesion may offer serious difficulty the well-circumscribed character of the translucent area within the lumen of the esophagus speaks definitely in favor of the benign nature of the lesion As the barium passing down the esophagus reaches the tumor there is a splitting of the stream a thin layer of barium passing between the tumor and the walls of the esophagus thereby encompassing the mass In the event that the tumor is small a thick suspension of barium may be objectionable in that it may actually obscure the lesion At times therefore a thin barium water suspension may aid in the visualization of such a lesion

2 When the tumor is intraluminal in position the radiologic appearance of the esophageal contour remains smooth and exhibits no evidence of irregularity An important point is the fact that the mucosal folds of the esophagus are intact Such evidence is of considerable value in differential diagnosis between a benign lesion and a malignant infiltration

3 If the tumor has a long pedicle there may be variation in the exact position of the tumor at different times and with change in the position of the patient Moreover in rare cases the pedicle of the tumor itself may be visualized as a translucent zone through the contrast medium in the esophagus This was well demonstrated in the carefully controlled case reported by Tamiya and Nosaki [15]

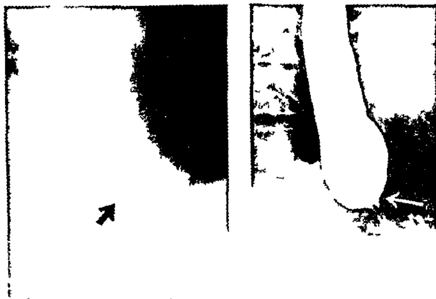


Fig 39 (A *Left*) Benign tumor of the esophagus Note the well rounded translucent area produced by the tumor within the confines of the distal end of the esophagus (B *Right*) Same patient as is shown in (A) The tumor is obscured by a large amount of barium Note also the dilatation of the esophagus apparently produced by occlusion of the lumen by the tumor

Because of the marked degree of mobility possessed by some of these benign tumors of the esophagus they may actually be brought up into the mouth as illustrated in the case reported by Samson and Zelman [16]

and in one case described by Weyrich, a pedunculated lipoma of the esophagus choked a patient to death by closing off the opening to the larynx [17]

4 The tumor may produce considerable dilatation of the esophagus when present at or near the cardiac end This occurred in the case reported by Haenisch [18] A mobile tumor attached by a pedicle by invaginating the cardiac end of the esophagus may be a cause of temporary obstruction at the cardiac end, with secondary dilatation In a case of this nature, roentgen examination at one time may exhibit a typical, rounded translucent area within an esophagus the lumen of which is normal, and re examination at a later time may show marked dilatation of the esophagus as a result of obstruction of the cardia Owing to this obstruction and the failure of the barium to make its exit into the stomach the barium suspension may fill up the dilated esophagus, thereby obscuring the tumor and arousing the suspicion of cardiospasm as a cause of the condition

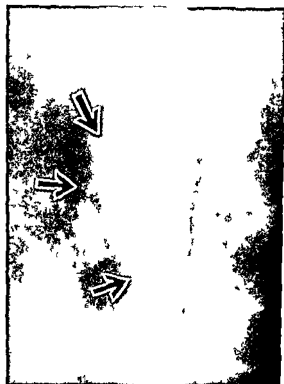


Fig 39 C Leiomyoma of the esophagus Note the fairly sharply defined concave defect of the anterior wall and the soft tissue shadow extending beyond the confines of the esophagus produced by the tumor

5 The determination of the point of origin of a benign tumor of the esophagus as to whether it is mucosal or submucosal, is extremely difficult and in many cases impossible One of the reasons is that, pathologically, a tumor originating within the wall of the esophagus may grow indirectly into the lumen and be indistinguishable from an intraluminal tumor of mucosal origin, certainly as far as the roentgen appearance is concerned When the tumor shows a rounded mass extending into the mediastinum away from the wall at the same time that it projects intraluminally one may entertain a strong suspicion that the mass is an intrinsic lesion of extra mucosal origin The fact that this mass moves with the changes in the position of the esophagus adds further support to this diagnosis However, extra esophageal masses which have become closely adherent to or have actually infiltrated into the wall of the esophagus may make differentiation from a primary lesion of the esophagus extremely difficult and often impossible

The serious technical difficulties involved are well demonstrated by the experimental efforts as well as the clinical observations of Schatzki and Hawes [19]

Differential Diagnosis In spite of all the difficulties that may arise the differentiation of a benign tumor from a malignant new growth depends primarily on the well circumscribed, intraluminal translucent area produced by the lesion with a smooth contour of the wall of the esophagus and an intact mucosa Mobility of this area also favors the diagnosis of a benign tumor attached by a pedicle

In malignancy of the esophagus, as will be discussed more fully later, the contour of the wall as a rule is irregular and rigid, the mucosal folds are destroyed, and a

proliferating mass if present extending into the lumen produces translucent areas frequently of a finger printing type and irregular in outline Moreover a mass of this nature is fixed and is not apt to exhibit evidence of change of position within the esophagus

Translucent areas within the esophagus may be caused by swallowed air trapped within the column of barium The differential diagnosis however, should offer no difficulty as translucent zones produced in this manner are of an inconstant character and may not be demonstrated on re examination Under fluoroscopic control the swallowed air may be noted passing through the course of the esophagus into the stomach

Illustrative Case The following is an example of a benign tumor of the esophagus

S F female aged 50 For about 8 years this patient had been complaining of attacks of substernal pain radiating to the back These attacks might occur at any time without any relation to meals At times the pain was extremely severe On one occasion the pain lasted throughout an entire night finally becoming so severe that she thought she was dying She vomited at times There was never a history of dysphagia Her weight fluctuated from time to time weight loss being only temporary and followed by gain in weight

Roentgen examination of the esophagus (Fig 39A) revealed a rounded translucent shadow at the cardiac end having the characteristics of a benign tumor Note also that Figure 39B shows the esophagus to be moderately dilated Owing to the large amount of barium present the tumor is obscured

Repeated esophagoscopic examinations revealed a polyp 36 cm from the upper incisor teeth and originating from the anterior wall of the esophagus A biopsy was done Histologic examination revealed no evidence of malignancy



Fig 39 (D E *Center* and F *Right*) Same patient as is shown in Figure 39 C Different views of the tumor removed at operation

The dilatation of the esophagus seen in Figure 39B may best be explained by a temporary mechanical blockage of the cardiac opening by the polyp This may also explain the clinical symptoms of recurrent attacks of severe substernal pain and vomiting

A benign intramural extra mucosal tumor of the esophagus is illustrated by the following fully controlled case

J S male aged 54 (patient of Dr Tolk) The patient gave a 3 months history of constant epigastric pain worse however, shortly after meals It was relieved by belching and

the application of heat In addition, he complained of a feeling in the midchest as if swallowed food could not go down He had lost 12 pounds

Physical examination was essentially negative

Roentgen examination of the esophagus (Fig 39C) revealed the following features

1 A fairly smooth localized, concave defect involving the midportion of the anterior wall

2 This area was observed to show slight changes in its contour in various films The posterior wall of this segment of the esophagus was entirely normal and peristalsis traversed this region

3 Of great importance was the fact that an elliptically shaped soft tissue shadow occupied the area of the concave defect

4 Particularly significant was the fact that this elliptical shadow extended beyond the confines of the lumen of the esophagus itself

Based on these findings the diagnosis was made of a benign submucosal tumor displacing the mucosa toward the opposite wall of the esophagus and at the same time extending beyond the confines of the esophagus itself It was also stated that this tumor would prove to be a leiomyoma of the midportion of the esophagus

The patient was operated on by Dr Nissen Through the left thoracic cavity the esophagus was exposed revealing a large hard tumor occupying its midportion approximately between the level of the bifurcation and the inferior border of the aortic arch The muscular layer of the esophagus overlying the tumor was incised It then became evident that the tumor did not infiltrate the muscular layer the submucosa or mucosa The tumor was dissected out leaving the mucous membrane intact The appearance of the tumor is shown in Figures 39 D E and F

Histologic examination of the tumor corroborated the preoperative diagnosis of leiomyoma

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9

Malignant Lesions of the Esophagus

ROENTGEN DIAGNOSIS

Carcinoma of the esophagus may be classified mainly according to two types The first is the scirrhous variety In its earliest stages this is submucosal in location causing a protrusion of the mucosa into the lumen Its gradual encroachment finally produces an annular stenosis At a later stage there may be invasion of the lumen and ulceration of the mucosa

In the second type there is an encroachment on the lumen of the esophagus by an expansive type of growth with an irregular cauliflowerlike contour and secondary ulceration Stenosis is produced by occlusion of the lumen Peristalsis is absent the wall becomes rigid and the lumen is narrowed There is a greater tendency in this type of malignant lesion for destructive changes to occur As the result of the breaking down of the tumor, there may be an increase in the lumen of the esophagus and a temporary alleviation of symptoms due to obstruction Malignancy of the esophagus remains localized for a long time

The carcinoma may also invade any of the neighboring structures either through extension of the growth or because of perforation The carcinoma may invade the wall of the trachea or the bronchi It may reach the cardiac area the pericardium, the pleura and the lungs The adventitia of the aorta may be infiltrated The vertebrae may be invaded and destroyed Tumefaction entering the vertebral canal may compress the spinal cord and lead to fatal paraplegia

Metastatic spread by way of the lymphatics and blood vessels may involve the lymph nodes the liver the lungs the kidneys the adrenals the pancreas and the bones

ROENTGEN DIAGNOSIS

A knowledge of the underlying pathology leads to a clearer understanding of the roentgen deformity of the esophagus produced by malignant lesions in this region When the infiltrative process is limited to the wall of the esophagus the contour is irregular the wall appears stiff peristalsis is absent and the lumen is narrowed The mucosa shows abnormalities as a result of the submucosal infiltration and secondary ulceration Proximal to the obstruction the esophagus becomes dilated In the proliferative type of malignant new growth the lumen becomes directly invaded the mucosa is destroyed and irregularly outlined translucent areas are present within it Almost invariably there is an associated irregularity in the contour of the involved region and the evidence of adenomatous invasion is associated with destructive changes produced by infiltration of the wall itself In such cases also the lesion leads to a narrowing of the lumen and dilatation of the esophagus proximal to the site of involvement

The dilatation of the esophagus however is never very great and never reaches the degree seen in those cases of obstruction produced by a lesion that is benign Two reasons explain this fact In the first place a patient with malignancy of the esophagus does not survive sufficiently long for extreme dilatation of the esophagus to take place The second reason is that owing to the breaking down of the tumor the ob

the application of heat In addition, he complained of a feeling in the midchest as if swallowed food could not go down He had lost 12 pounds

Physical examination was essentially negative

Roentgen examination of the esophagus (Fig 39C) revealed the following features

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assume that the cause is due to cardio spasm

Considerably greater difficulty in differential diagnosis may arise in some cases when the stenosis of the esophagus is produced by the action of a corrosive. While the effect of the corrosive may be such as to produce an irregularity involving almost the entire length of the esophagus and therefore readily recognizable as having been produced in this manner a localized





Fig 40 (*Left*) Carcinoma at the pharyngo esophageal junction
Fig 41 (*Right*) Carcinoma of the esophagus

area of destruction and irregular narrowing may closely simulate the roentgen appearance of a deformity produced by a new growth In such borderline cases the clinical history will be the decisive factor in differentiating the two conditions

The appearance of the esophagus produced by other organic lesions of an intrinsic nature will be discussed in later chapters

Illustrative Cases A carcinoma may be present at the mouth of the esophagus

S F male aged 66 The patient gave a history of dysphagia of 18 months duration ultimately he had difficulty in swallowing even liquids He had lost considerable weight He stated that at the onset of his distress a lump had been removed from the left lower cervical region and that examination showed this to be malignant

Physical examination revealed a pallid white male, showing evidence of recent weight loss An operative scar was present in the left lower cervical region The examination was otherwise essentially negative Endoscopy revealed the cricopharyngeus region to be friable and hard A biopsy was taken from the area of apparent tumefaction

The pathologic diagnosis was squamous cell carcinoma of the esophagus

Roentgen examination (Fig 40) revealed complete obstruction to the flow of barium at about the junction of the hypopharynx and the esophagus with a cone shaped defect

The roentgen diagnosis then was that a mass was causing obstruction at the pharyngo esophageal junction Pathologic examination as noted above showed the tumor to be a squamous-cell carcinoma

The next case is that of a carcinoma of the proximal portion of the esophagus

A W male aged 52 The patient had complained of increasing dysphagia for 2 months Prior to that he had complained of a

struction is usually incomplete When the *obstruction is due to a benign lesion*, however, as in cardiospasm, the condition may progress for many years Moreover, the obstruction is more complete and no canalization develops, as in the breaking down of the tissue of a malignant tumor

It is for this reason also that the degree of dilatation of the esophagus and the evidence of obstruction may show considerable diminution at times in the case of a malignant lesion The sloughing of a large mass may eliminate the cause of obstruction

While, as a rule, malignant lesions of the esophagus are single, in rare instances two malignant lesions may be present, with evidence of normal intervening tissue as determined both roentgenologically and on pathologic examination

Another important roentgen finding is due to the fact that malignant lesions of the esophagus may perforate neighboring tissue *and particularly a bronchus Under such* conditions an irregularly outlined barium shadow will be seen extending beyond the confines of the growth itself for a variable distance In those cases in which the bronchus has been perforated by the growth, barium will be noted making its exit through the fistulous communication and outlining the tracheo bronchial tree In this connection it is important to remember that as a result of the stenosis produced by the new growth particularly when this is high in location there may be *a regurgitation of barium up into the* pharynx and then into the trachea and the bronchi Unless the patient has been fluoroscoped with considerable care the findings may be misinterpreted as being the result of an actual fistulous communication between the esophagus and the trachea or the bronchus By observing the behavior of the esophagus as the patient drinks the barium, it may be possible to determine which of the two factors is at fault When a fistula is present, the actual exit of the barium from the new growth may be noted Because of the danger that the patient with a stenosing malignant lesion may regurgi*tate the barium* during examination, it is important that only a small amount be administered A teaspoonful of the barium acacia mixture may be sufficient and, as *previously emphasized if clinical evidence* of stenosis is marked a thinner barium water mixture, possibly only slightly thickened with acacia, may be employed

In some cases, it is possible for a tracheo esophageal fistula to be present and yet go unrecognized roentgenologically Either the area of communication may be extremely small or it may be so walled off by edema and inflammation as to defy detection

Differential Diagnosis The differential diagnosis between a benign and a malignant tumor has been previously considered in the discussion of benign tumors of the esophagus

The differentiation from cardiospasm should ordinarily offer no difficulties *Malignant invasion may occur almost any*where in the course of the esophagus although the site of predilection is more apt to be at the points of normal constriction In cardiospasm the lesion is limited to the cardiac end of the esophagus Limitation of a malignant infiltration to the cardiac end of the esophagus does occur but such localization is not common, except as an extension from a carcinoma of the cardiac end of the stomach In malignancy there is irregularity of contour and destruction of normal mucosal folds In cardiospasm the *contour is extremely smooth and tapers to* a fine line Food retention may produce irregularity of contour in cardiospasm and this is one reason why the patient being examined should have had no food for at least 12 hours Re examination of the patient in such cases if he is properly prepared will show that the irregularity in cardiospasm is no longer present

Moreover, as already stated, the degree of dilatation of the esophagus in cardiospasm may be enormous In new growth this does not become nearly so marked If the dilatation is extreme one may safely

The patient was markedly emaciated The left pupil was larger than the right and both were of irregular contour There was a feeling of resistance due to a mass deep in the neck

Esophagoscopy showed a large fungating mass in the proximal portion of the esophagus *The diagnosis on microscopic examination* was squamous-cell carcinoma

Roentgen examination (Fig 42) revealed evidence of an annular malignant lesion with marked obstruction involving the proximal portion of the esophagus about 1 inch distal to the pharyngo-esophageal junction As a result of the obstruction the entire pharynx was distended with barium Note that the esophagus proximal to the area of narrowing is distended in such a way that it simulates the appearance of a diverticulum

The following is an example of carcinoma of the middle third of the esophagus

J M male aged 58 This patient gave a 4 months history of difficulty in swallowing this gradually increased in severity and eventually became very painful Pain was retrosternal and radiated down to the epigastrium Occasionally he vomited the food just eaten He had lost 40 pounds Esophagoscopic examination revealed the following The esophagoscope was passed to a point 29 cm from the upper margin A fungating soft mass was seen obstructing the lumen A small *bougie was passed below this obstruction* A specimen was removed for biopsy The pathological diagnosis was squamous-cell carcinoma of the esophagus

Roentgen examination (Fig 43) revealed a diffuse narrowing of approximately the middle third of the esophagus with irregularly outlined translucent areas within this region There was moderate obstruction of the esophagus above the area of involvement The roentgen diagnosis was carcinoma of the esophagus

Less commonly a carcinoma of the esophagus may be limited to the cardiac end, as in the following case

J G male aged 63 This patient complained of the sudden onset of cramps in the epigastrium 7 weeks before admission to the hospital At the time of admission the pain was continuous and was aggravated by eating Vomiting had continued until 2 weeks before He had extreme difficulty in swallowing and could retain only water He lost 15 pounds in 2 months

At operation a hard annular tumor surrounding the cardiac portion of the stomach was found It extended downward from the diaphragmatic opening for a distance of about 6 cm along the circumference of the stomach There were many small nodular white masses in the liver evidently secondary deposits from this growth

Roentgen examination of the esophagus (Fig 44) revealed a moderate degree of obstruction at its junction with the cardiac end of the stomach There was considerable irregularity of contour at the cardiac end which is characteristic of new growth



Fig 44 Carcinoma of the cardiac end of the esophagus

Rarely multiple malignant lesions of the esophagus may occur The following is an example of this

F W male aged 49 About 6 weeks prior to his admission to the hospital this patient had begun hiccoughing at about 2 P M , continuing for about 2 hours This had recurred almost every afternoon since the onset It was occasionally relieved by drinking from 2 to 3 glasses of water The patient lost 36 pounds in 2 months Esophagoscopy revealed a definite annular constriction about 12 cm below the cricopharyngeus muscle The area was pale and friable and it bled easily Gastrostomy re

productive cough, occasional night sweats and weakness

Roentgen examination of the chest showed chronic pulmonary tuberculosis

Esophagoscopy showed a new growth 20 cm from the teeth The growth was irregular, middle thirds, with destruction of the normal mucosal markings, classical evidence of a new growth

When the carcinoma of the esophagus is high in location the pharynx may be dis





Fig 42 (*Left*) Carcinoma of the esophagus

Fig 43 (*Right*) Carcinoma of the esophagus Note the finger printing' deformity

firm cauliflowerlike in appearance bleeding readily to the touch Biopsy showed the lesion to be a squamous cell carcinoma

Exploratory thoracotomy revealed the following facts From the aortic arch upward the periesophageal tissues were invaded and the esophagus was fixed by inflammatory and probably neoplastic tissue The hilus of the left lung was also adherent in this area The condition was regarded as inoperable and the chest was closed without drainage

Roentgen examination of the esophagus (Fig 41) revealed an irregular annular narrowing at the junction of the upper and the

tended with barium This is illustrated by the following case

L L male, aged 67 About 2 months before his admission to the hospital this patient complained of the feeling of a lump in his throat on swallowing He described the lump as being at the level of the cricoid cartilage He had increasing difficulty in swallowing Finally 3 weeks before admission he was unable to swallow even liquids in any but minute amounts The swallowed food was regurgitated almost at once He had lost 30 pounds in the preceding 3 weeks

vealed that the stomach was markedly dilated There were firm nodular masses extending along the lesser curvature of the stomach to the duodenum

The autopsy revealed the following Opposite the bifurcation of the trachea on that surface of the esophagus there is a large ulcerated indurated necrotic lesion about 7 cm by 4 cm in size The ulcerative process has extended to but not into the trachea and aorta All but a small portion of the circumference of the esophagus is involved in this lesion A similar lesion involves the esophagus at the cardiac orifice of the stomach and appears to have extended down into the fundus of the stomach, at which point it is perforated posteriorly into the lesser peritoneal cavity

Microscopic examination of the esophageal tumor showed ulceration of the epithelium and invasion of the muscularis with tumor cells singly and in masses The cells were polyhedral variable in size and granular The nuclei were large oval or circular Mitotic figures were fairly common

The diagnosis was transitional-cell epithelioma

Roentgen examination revealed an irregularly outlined markedly narrowed area of the esophagus involving the middle third There was considerable dilatation of the esophagus above this region (Fig 45A) The lower third of the esophagus again assumed a normal appearance as far as the cardiac end In this region there was evidence of neoplastic infiltration Particularly significant was a translucent area at the extreme cardiac end of the esophagus encompassed on either side by a thin stream of barium, which was best seen



FIG 48 (A *Left*) Malignancy of the esophagus with tracheo esophageal fistula showing at first examination (B *Right*) There is no evidence of the tracheoesophageal fistula at this later examination although the fistulous communication was found subsequently at autopsy





Fig 45 (A *Left*) Carcinoma of the esophagus
Fig 45 (B *Right*) Same patient as is shown in Figure 45 A Note the independent new growth at the cardiac end of the esophagus





Fig 46 (*Left*) Carcinoma of the esophagus with fistula
Fig 47 (*Right*) Carcinoma of the esophagus with tracheo esophageal fistula

cation was temporarily occluded in some manner possibly by inflammatory pathology or by a particle of food which had become lodged at the fistula

A rare type of malignant infiltration of the esophagus is that produced by secondary invasion from a primary lesion external to the esophagus The following is a case of this nature

J R male aged 29 For 3 years this patient had complained of a lump in the left side of his neck which gradually became considerably enlarged There was no discharge Caseous material was obtained on aspiration Esophagoscopy and bronchoscopy revealed constriction of the trachea and of the lumen of the esophagus A biopsy taken from a small mass in the esophagus revealed a transitional cell sarcoma which was considered to be secondary to a malignant tumor of the thymus Following a course of roentgen and radium therapy the size of the mass which previously had grown from the left side across the suprasternal space to involve the right base of the neck became considerably reduced in size Partial resection of the mass was carried out It was found to extend from underneath the left lobe of the thyroid down to the anterior mediastinum The pathologic report of the resected mass was transitional cell epithelioma The wound healed but left a few draining sinuses A small mass developed in the left supraclavicular region which appeared like an abscess and yielded a large amount of purulent material after being incised The next day air and gastric contents passed through the sinus Apparently this was an esophageal tracheal fistulous sinus

Roentgen examination of the esophagus (Fig 49) revealed marked infiltration of the upper portion with numerous irregularly outlined translucent areas apparently secondary to the invasion of the tumor of the thymus

As a rule malignancy of the esophagus is of primary origin Metastatic spread to the esophagus from a primary carcinoma in a distant location is very rare Von Recklinghausen[1] described a myxochondrosarcoma of the tibia which showed generalized metastases associated with implantation in the esophagus Spiegelberg s case was a melanosarcoma of the left eyeball followed by death 13 months after enucleation In addition to generalized metastases the autopsy disclosed implantations in the esophagus

Gross and Freedman[3] described a case of an obstructing tumor of the esophagus which was due to a metastatic implantation from a primary carcinoma of the prostate

Toreson[4] described the findings in three patients autopsied at the Pathological Institute of McGill University in which metastatic carcinoma of the esophagus was found originating in a primary lesion elsewhere In two cases the primary lesion was a carcinoma of the breast in the third case a histoid teratoma with embryonal carcinoma of the testis All three patients had developed dysphagia before death and the autopsies showed that metastatic lesions had occluded the esophagus

Differential diagnosis on the basis of the roentgen evidence between a primary lesion and one of metastatic origin cannot be made

Rarely a carcinoma may develop at the site of a benign stricture of the esophagus Benedict in a survey of the literature found reports of 31 cases of carcinoma developing at the site of an originally benign stricture of the esophagus To these he added 2 cases of his own Of all these case 16 occurred in strictures which had resulted from lye

Sarcoma of the Esophagus Sarcoma of the esophagus is rare Chapman in 1877[6] reported the first case of a tumor which had occluded the esophagus and which at autopsy proved to be an oval and spindle celled sarcoma The spindle celled sarcoma reported by Targett in 1888[7] is preserved in Guy s Hospital Museum and is the first example of this type of lesion recorded in the transactions of the Pathological Society of London Although the number of reported cases has gradually increased sarcoma of the esophagus may still be considered a very rare disease The most unusual type is the leiomyo sarcoma first reported by Howard[8]

As already noted (page 62) sarcoma of the esophagus may simulate the appear

in the examination of the stomach (Fig 45B), indicating a second tumor

A serious complication of malignancy of the esophagus is the development of a fistula into the periesophageal tissues (Fig 46) This may ultimately perforate into adjacent structures, particularly into the trachea or bronchus (Fig 47)

A fistulous communication between a malignancy of the esophagus may be present and yet may not always be demonstrable roentgenologically This is seen in the following example



Fig 49 Marked irregular infiltration of the upper portion of the esophagus produced by a tumor of the thymus

About 6 months before his admission to the hospital this patient for the first time had had difficulty in swallowing solid food Swallowing was accompanied by pain in the chest Occasionally he had difficulty with liquids as well He vomited on several occasions but the vomitus was never bloody He lost 15 pounds in 6 weeks His temperature was 101° F

Esophagoscopic examination revealed a carcinoma involving the bronchi and the esophagus At the point of the fistula, the lumen was narrowed A gastrostomy was done

The autopsy revealed the following About 7 cm below the epiglottis the mucous membrane of the esophagus is discolored and ulcerated and the tissues are inelastic and hard Carcinomatous change is rather extensive encircles the esophagus completely and extends over an area distally of from 6 to 7 cm Crypts and pockets have been formed in the esophageal wall irregularly throughout the tumor The lumen of the esophagus is only moderately constricted over the affected zone Near the lower portion of the tumor is a perforation having diameters of 1 cm by 0 5 cm which communicates directly with the trachea about 5 cm above the tracheal bifurcation At the point of perforation, as well as throughout the tumor zone the trachea is densely fused to the esophageal wall the adhesed area being stone hard and inelastic

"The microscopic examination of the esophagus showed the squamous cell epithelial lining to be missing in many places Where it is present it is orderly and regular The subepithelial stroma is greatly thickened and densely infiltrated with lymphocytes fibroblasts and plasma cells in several areas the squamous cell lining is replaced by masses of amorphous necrotic tissue, throughout which no malignant tissue can be distinguished The diagnosis is chronic inflammatory tissue with necrosis (post radiation) Cervical lymph nodes The lymphoid tissue is generally sparse throughout and the fibrous tissue prominent Several zones are seen where the lymph structures are completely replaced by nests of invading epithelial cells which appear definitely squamous in character This indicates metastatic carcinoma

The tracheo esophageal fistula was evidently the result of the perforation of a squamous cell carcinoma of the esophagus

Radiographic examination (Fig 48A) revealed an area of malignant infiltration at the junction of the upper and middle thirds of the esophagus with barium outlining the trachea at about the site of the infiltration It was interesting to note that a roentgen examination of the esophagus made 5 days later (Fig 48B) failed to show any barium within the trachea It was therefore believed that the appearance at the time of the first examination was the result of regurgitation of barium from the esophagus by way of the pharynx as a result of the difficulty in swallowing However as shown on the esophagoscopic examination and on autopsy, an actual esophageal tracheal fistula existed One must assume therefore that at the time of the second radiographic study the zone of fistulous communi

10

Foreign Bodies in the Esophagus

ROENTGEN DIAGNOSIS

According to Palugyay, Hochenegg was the first (1896) to demonstrate the value of localization of foreign bodies in the esophagus by means of the roentgen ray He also employed fluoroscopic visualization as a guide in the removal of such foreign bodies

Iglauer and Ransohoff[1] first called attention to the presence of air in the cervical region at the site of perforation of the esophagus by a foreign body Iglauer suggested the administration of a small quantity of lipiodol A streak of oil beyond the confines of the esophagus itself speaks for perforation

McGibbon and Mather[3] reported two cases in which, as a result of the perforation there was a collection of air which displaced the esophagus anteriorly Both the presence of the air as well as the displacement of the esophagus produced by it could be demonstrated in the roentgenogram The depth between cervical vertebrae and trachea is increased





Fig 51 (*Left*) Cup-shaped defect due to obstruction of the esophagus by food

Fig 52 (*Right*) A rounded translucent intraluminal shadow produced by a piece of lamp chop

Nonopaque foreign bodies commonly encountered are chicken bones, buttons of a

ance grossly and roentgenologically of a benign, intraluminal, polypoid tumor and even be possessed of a pedicle In many cases however the deformity produced by the lesion is indistinguishable from that of carcinoma

The following is a case of sarcoma of the esophagus



Fig 50 Fibrosarcoma of the esophagus

Illustrative Case P F male, aged 66 The patient gave a 7 months history of dysphagia at first limited to solid foods but later extending even to liquids He had lost 45 pounds during this time

Esophagoscopy revealed a sloughing mass on the posterior wall involving both lateral walls of the esophagus about the middle third There was almost complete obstruction of the lumen

Pathologic examination of a biopsy specimen removed through the esophagoscope was reported as follows "Microscopic examination shows pieces of tissue composed of a tumor mass which consists of oval round and spindle shaped cells the latter predominating The cells show different staining properties the majority are hyperchromatic The tumor cells are embedded in a connective tissue stroma which contains numerous giant cells and many mitotic figures and eosinophiles Diagnosis fibrosarcoma "

Roentgen examination (Fig 50) showed a marked, irregular narrowing of the esophagus at the junction of the upper and middle thirds with almost complete obstruction in this region and secondary dilatation above the area of involvement

From the roentgen appearance one could state only that a malignant lesion was present and therefore in all probability that the pathologic process was a carcinoma There are no features in the behavior of this lesion which might have led to the suspicion that the tumor was a sarcoma The diagnosis depended entirely upon the histologic characteristics of the new growth

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prompt recovery and within 3 days was able to swallow food

Roentgen examination (Fig 51) revealed a translucent area at about the midportion of the esophagus with a cup shaped, rounded defect intraluminal in position preventing the barium above it from passing distally The smooth rounded intraluminal character of the shadow is characteristic of a foreign body causing obstruction

Figure 52 shows the irregularly mottled translucent area produced by a piece of lamb chop stuck in the esophagus

Almost any object may be stuck in the esophagus Figure 53 represents a safety pin An unusual example of a foreign body is shown in Figure 54, a sword introduced by the patient and occupying the entire length of the esophagus

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Fig 53 Safety pin in the esophagus

radiolucent character or pieces of meat They are usually lodged at the suprasternal notch In some cases a splitting of the stream may be noted with a thick suspension of barium sulfate in water or if the occlusion is marked there may be an arrest of the flow of barium After most of the barium has left the esophagus in those cases in which obstruction is only slight there may be a residual fleck of barium sticking to the foreign body Manges[4] was able to demonstrate the thread holes in buttons after administering barium by the distribution of the barium over them

Scott and Moore recommended a very thin suspension of barium in water so as to considerably diminish the opacity of the esophagus after complete filling Through this thin layer, the filling defect produced by the foreign body may be seen

A foreign body sometimes may be demonstrable after the administration of a barium containing capsule, which is arrested at the site of obstruction

Illustrative Case An example of the deformity produced by the impaction of food within the esophagus is shown in the following case

J C, male aged 55 This patient had a stricture of the esophagus as the result of having accidentally swallowed a solution containing lye 6 years before his admission to the hospital Gastrostomy was done because of the esophageal stricture He had been dilating this stricture of the esophagus for 2 years preceding admission Two days before admission, he found that he was unable to dilate the stricture and was unable to swallow food This occurred following a meal The patient said that he had had in the past similar attacks of difficulty in swallowing as a result of food having become lodged in the esophagus above the stricture This patient made a



Fig 54 Sword in the esophagus

of ascites or positive liver function tests In the case of a tumor of doubtful origin in the region of the spleen the demonstration of varices in the esophagus would indicate that the tumor is actually splenic in origin The demonstration of varices may also explain the cause of hemorrhage in an individual exhibiting no frank evidence of disease and in whom examination of the stomach and intestines is roentgenologically negative A knowledge of the presence of such varices will also guard against the indiscriminate u e of the stomach tube



FIG 56 (A *Left*) Esophageal varices Note the beadlike translucencies in the mid portion (B, *Center*) Same patient as is shown in (A) Note the variation in the appearance produced by the varices (C *Right*) Same patient as is shown in (A) Autopsy specimen Note the probe inserted into the opening in the vein from which fatal bleeding occurred

Essential Roentgen Findings The essential findings in the roentgen demonstration of esophageal varices are therefore

1 The presence of rounded, translucent areas beadlike in appearance and usually involving the distal portion of the esophagus

2 There may be momentary variation in the appearance of these areas primarily as a result of peristaltic activity that being the most important factor beyond the control of the examining physician

As a rule there should be no difficulty in differential diagnosis Trapped globules of air can be readily recognized by their inconstancy and because of the fact that their continuous change in position and their final entrance into the stomach may be noted during fluoroscopic examination

Diagnostic differentiation from a malignancy should as a rule offer no serious difficulty In the presence of esophageal varices peristalsis is intact and the rounded translucent areas show variations in prominence in different films as a result of those factors which have already been considered In malignancy the contour is irregular, rigid and free of peristaltic activity Any invasion of the lumen by a proliferating new growth produces irregularly outlined translucent areas ordinarily lacking the

actual mechanical presence of the varices themselves, which partially occlude the lumen

A comparison of different exposures made in rapid succession may show considerable variation in the appearance produced by the varices Such inconstancy of appearance, far from indicating that no real organic disease is present, is evidence that actually favors the diagnosis of varices for the following reasons The presence or absence of these translucent, beadlike areas depends on the degree of engorgement of the blood vessels at the time of examination This will be influenced by the peristaltic activity of the esophagus Small varices may be completely stripped of their blood during peristalsis and therefore may fail to show at that particular moment In later stages, peristalsis may strip only the varices in the upper portion of the esophagus As the blood enters the lower varices, these may become markedly distended and may show in an even more exaggerated form Considerable change in the roentgen appearance of esophageal varices may, therefore, take place in different observations over a period of a few seconds

As already noted, the degree of distention of the thoracic veins will vary with the respiratory phase, so that roentgenograms taken during inspiration will show more pronounced manifestations of their presence If exposures have also been made during expiration, this will be a factor in causing a variation in the relative prominence of the roentgen findings And, as previously stated, the ingestion of a large amount of barium may also force blood from the varices and therefore be another factor in causing variations in their relative prominence



Fig 55 (A, *Left*) Esophageal varices (B, *Left center*) Same patient as is shown in (A) Note the variation in the appearance produced by the varices (C *Right center*) Same patient as is shown in (A) Note further variation in the appearance produced by the varices (D *Right*) Same patient as is shown in (A) Note that the appearance produced by the varices is different in each observation This is characteristic of the deformity produced by esophageal varices

In some cases, an examination may be made for suspected varices after hemorrhage has occurred Failure to find roentgenologic evidence at this time does not rule out their presence, but may mean that the exsanguinating process has emptied the varices in such a way that they no longer protrude into the lumen, with the production of their characteristic pattern of the relief picture

Clinically, the demonstration of esophageal varices may be of considerable significance They may be demonstrable at a time when cirrhosis of the liver cannot be shown in any other way, either by the presence

on five occasions He again had a recurrence of vomiting of blood the following year One week before admission he developed fluid in the abdomen requiring paracentesis About 3 quarts of fluid were removed His abdomen however continued to distend because of the prompt reaccumulation of fluid He had had a suprapubic cystostomy for carcinoma of the prostate 6 years before



Fig 58 (A *Left*) Esophageal varices (B *Right*) Same patient as is shown in (A) Note the changes in the pattern produced by the varices Note also the transition from normal mucosal folds to the distortion produced by the varices

Physical examination revealed marked abdominal distention due to fluid and a suprapubic tube draining the bladder On rectal examination there was a large nontender irregular nodular prostate The patient vomited blood on a few occasions while in the hospital and died in spite of transfusion

Autopsy revealed a carcinoma of the prostate with urethral obstruction and a portal cirrhosis of the liver

With regard to the gastro intestinal tract the report was as follows The esophagus presents in its lowermost end several tortuous

beadlike arrangement of varices Moreover, the appearance is identical in the different films and is not influenced either by peristaltic activity or by variations in the respiratory phase during which exposures have been made

Illustrative Cases J T male, aged 31 During the previous 1½ days before admission, this patient had been vomiting blood dence of varices in the distal third of the esophagus

Roentgenographic examination (Fig 55A B C D) revealed numerous irregularly rounded, translucent areas intraluminal in location and primarily limited to the cardiac end of the esophagus The various films show a marked alteration in the mucosal relief picture due to the momentary changes in the degree of distention of the varices At times the translucent areas appear as if outlining

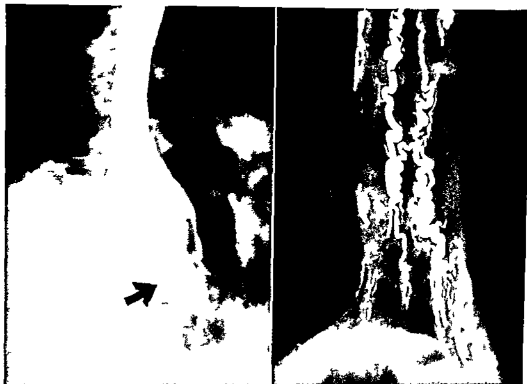


Fig 57 (A *Left*) Esophageal varices Note the translucent areas at the cardiac end (B *Right*) Same patient as is shown in (A) Roentgen appearance of the esophageal veins in the autopsy specimen after injection with barium suspension

and passing tarry stools In addition he had had pain which was sudden in onset and present in the epigastrium and periumbilical region It was colicky in nature He had had no previous episodes of this kind

Physical examination revealed the presence of ascites The spleen was markedly enlarged The liver was not palpable There was a severe hypochromic anemia The spleen was particularly palpable after the ascites had diminished under treatment with ammonium chloride and mercupurin It was then palpable five fingers breath below the left costal margin

Esophagoscopic examination showed evi

a tortuous vein in the course of which may be noted small rounded beadlike protuberances At times also, some of the translucent areas appear as sharply punched out defects of contour

Variations in the mucosal relief of the esophagus are also illustrated by the following case

D C male, aged 70 Three years previously the patient had been admitted to the hospital after having vomited a considerable amount of blood At that time he gave a history of having had abdominal paracentesis

on five occasions He again had a recurrence of vomiting of blood the following year One week before admission, he developed fluid in the abdomen requiring paracentesis About 3 quarts of fluid were removed His abdomen however continued to distend because of the prompt reaccumulation of fluid He had had a suprapubic cystostomy for carcinoma of the prostate 6 years before

Physical examination revealed marked abdominal distention due to fluid and a suprapubic tube draining the bladder On rectal examination there was a large nontender irregular nodular prostate The patient vomited blood on a few occasions while in the hospital and died in spite of transfusion



Fig 58 (A *Left*) Esophageal varices (B *Right*) Same patient as is shown in (A) Note the changes in the pattern produced by the varices Note also the transition from normal mucosal folds to the distortion produced by the varices

Autopsy revealed a carcinoma of the prostate with urethral obstruction and a portal cirrhosis of the liver

With regard to the gastro intestinal tract the report was as follows The esophagus presents in its lowermost end several tortuous

elevated, linear areas due to underlying varicose veins At several small points small blood clots are noted and pressure on these dilated veins displaces the blood clot and forces blood into the esophagus "

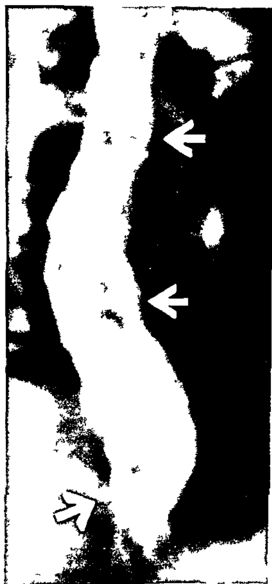


FIG 59 Esophageal varices Note the dilatation of the esophagus as well as the translucent areas produced by the varices

During his final brief hospitalization of 8 days before death, this patient s physical condition precluded any roentgen studies However, roentgen examination of his esophagus had been made 3 years before this last admission

Figure 56A showed a superficial irregularity in the contour of the distal half, with an elongated translucent area hugging the anterior wall At about its midportion are several rounded, beadlike translucencies and proximally an elongated translucent area Figure 56B showed the appearance of the esophagus at another stage when the distal portion was narrowed and an elongated, centrally located translucent zone was present in this region The rounded beadlike translucent areas previously described were again present although there was a slight change in their configuration Proximal to this region was a vaguely outlined, elongated, centrally located translucent zone with very small, rounded areas in its course The entire picture was that of esophageal varices and explained the clinical symptomatology the ascites and the vomiting of blood Figure 56C showed the appearance at autopsy with a probe through an opening in a distended esophageal vein from which the bleeding had occurred

Short elongated areas of translucency with deformity of the contour produced by varices is illustrated by Figure 57A in the case of S V These findings were transient, however, and there was considerable variation during the course of examination The diagnosis of varices was controlled by autopsy findings Figure 57B showed the roentgen appearance of the esophageal veins in the autopsy specimen after injection with a barium suspension

The transition from normal mucosal folds to the distortion produced by varices is illustrated by the next case

M L female aged 52 The patient gave a 5 weeks history of generalized abdominal discomfort vomiting and moderate weight loss During the preceding 5 days she noticed a swelling of the abdomen Physical examination revealed evidence of ascites enlargement of the liver engorgement of the cervical veins and moderate ankle edema She had severe secondary anemia

The roentgenographic examination (Fig 58A and B) showed classical changes in

the distal half of the esophagus produced by esophageal varices Figure 58A showed a beadlike arrangement along the course of the distal portion of the esophagus near its anterior wall In Figure 58B considerable change in the deformity is seen a characteristic of esophageal varices Rounded translucent areas not shown in Figure 58A may be noted in Figure 58B probably due to the thinner layer of barium Note also by way of contrast the normal parallel arrangement of the mucosal folds in the proximal portion of the esophagus in Figure 58B

The esophagus may become considerably dilated because of varices as in the illustrative case that follows

L F, male, aged 5? The patient gave a history of lues and of pulmonary tuberculosis Three days before admission to the hospital he developed epigastric pain which was shortly followed by the vomiting of blood and by tarry stools He had had no previous gastro intestinal symptoms While under observation his abdomen became distended as a result of ascites

The autopsy revealed portal cirrhosis of the liver in addition to pulmonary tuberculosis There were marked esophageal varices which extended all the way up to the pharyngeal region

The roentgenographic examination (Fig 59) showed marked dilatation of the entire esophagus with intraluminal translucencies throughout its entire length There was considerable change in the appearance of these areas in the different films No other cause but varices was found at autopsy to explain the dilatation The widespread character of the involvement was also confirmed at autopsy, which showed varices as high up as the pharyngeal region

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12

Peptic Ulcer of the Esophagus

ROENTGEN DIAGNOSIS

Among the earliest contributions to the study of peptic ulcer of the esophagus was that of Quincke,[1] who in 1879 described three cases These lesions were present in the distal portion of the esophagus and, in his opinion, had been produced by the action of regurgitant gastric juice In his third case stenosis resulted from the action of the ulcer

The condition had been considered extremely rare by Rokitansky and its existence had been completely denied by no less an observer than Birch Hirschfeld Quincke, however dissipated all doubt by his histologic studies

At the time of the publication of his paper in 1906 Tileston found records of 41 cases of peptic ulcer of the esophagus described in the literature To these cases Tileston added his observations in 3 cases of his own A study of the records of the Massachusetts General the Boston City and the Long Island Hospitals disclosed 6 cases of peptic ulcer of the esophagus in a series of 4 496 autopsies an incidence of 0 13 per cent In the Bellevue Hospital series of 22,810 autopsies from 1905 through 1935, there were 30 cases of ulcer of the esophagus or an incidence of 0 13 per cent Strangely enough, this figure is identical with that of Tileston The probable accuracy of this relative incidence is enhanced by the fact that it holds for the much larger autopsy series at Bellevue Hospital

Ewald[3] in 1910 described stenosis of the esophagus resulting from the presence of a peptic ulcer

Another important contribution to the study of peptic ulcer of the esophagus was that of Cantieri [4] In his monograph he made a thorough survey of the literature up to 1910 and credited Wolcamero with having recorded the first case in 1671 Cantieri collected reports of 62 cases and added 6 of his own including the actual pictures of the specimens showing the presence of the ulcer of the esophagus

Miller in 1912[5] described an ulcer of the esophagus just above the cardiac orifice about 1⅓ inches in its greatest diameter, which had perforated into the left mediastinum and left pleura A duodenal ulcer was also present

Watson in 1912[6] described two cases of ulcer of the esophagus found at autopsy at St Bartholomew s Hospital In both cases, the ulcer was situated immediately above the cardiac orifice One had perforated into the left pleura Perforation of two ulcers at the lower end of the esophagus found at autopsy in a patient who died of bronchopneumonia was reported by Gott [7]

Simple ulcer of the esophagus resembles that of the stomach both grossly and microscopically It is usually found just above the cardia Ulcers vary greatly in size ranging from those the size of a pea or smaller to those involving the entire surface from the bifurcation to the cardia As a rule only one ulcer is present rarely, the condition is multiple The small ulcer may involve only the mucosa, or in the more chronic and older form it may have the typical punched out appearance of a gastric ulcer There is a great tendency to perforation Healing may take place with scar formation, with puckering and radiation or with actual stenosis

A significant factor which may explain the etiology of such peptic ulcers is the presence of aberrant gastric mucosa in the wall of the esophagus This was demonstrated by Johanna Hellmann,[8] Taylor,[9] Jackson[10] and Stewart and Hartfall[11]

Kelly[1] examined the esophagus histologically in 17 children under 1 year of age and found islets of gastric mucosa in some of these cases In one infant 10 weeks old such misplaced gastric mucosa was found in association with an esophageal ulcer just above the level of the diaphragm There were marked chronic inflammatory changes with early fibrosis as far up as the bifurcation of the trachea

Rector and Connerley[13] examined the esophagus in 1 000 consecutive autopsies of infants and children and found aberrant gastric mucosa in 118 cases Inflammation with and without ulceration was frequently present in this heterotopic mucosa An interesting observation which they made is that in 51 per cent the aberrant gastric mucosa was in the upper third of the esophagus, in 41 per cent in the middle third and in only 8 per cent was it present in the lower third Acid gastric secretion from such aberrant glands may therefore underlie the formation of the full blown ulcer

Another factor may be a retrograde flow of gastric juice up into the distal portion of the esophagus In some cases peptic ulcer has been noted at the junction of a congenitally short esophagus with a thoracic stomach The acid gastric secretion within the thoracic stomach by regurgitation may be a definite factor in the explanation of such esophageal ulceration

A rare cause of peptic ulcer of the esophagus is cerebral or cerebellar disease Cushing[14] described the case of a child of 10 operated on for a cerebellar tumor At autopsy there was a perforated lesion in the esophagus about 3 cm in length In another case following extirpation of a right parietal metastatic hypernephroma autopsy disclosed both gastric and esophageal perforation

Evidence of perforation of the esophagus as noted at autopsy in a patient suffering from acute encephalitis was described by Masten and Bunts[15]

ROENTGEN DIAGNOSIS

The outstanding feature in the roentgen diagnosis of peptic ulcer of the esophagus is the demonstration of the niche Associated with this there is usually an area of stenosis at the site of the niche The lesion usually involves the distal portion of the esophagus

Apparently the first roentgen demonstration of peptic ulcer of the esophagus was that by Brunetti[16] The diagnosis was based on the presence of a large niche in the lower third of the esophagus This was accompanied by spasm of the esophagus in the region of the niche during the swallowing of a bolus of food There was a history of recurrent hemorrhage although no lesion other than the one in the esophagus was demonstrable The patient apparently died of perforation Unfortunately no autopsy confirmation was obtained

Fleischner[17] described a case in a man 64 years of age who gave a 3 year history suggesting gastric ulcer Roentgen examination showed two niches of the esophagus just above the diaphragm The longitudinal folds radiated towards them About ½ year later an irregularly outlined sacculation was present, 5 mm by 15 mm in size at the location of the previous niches The patient at this time complained of vomiting and of pain upon ingestion of liquid as well as of solid food A gastrostomy was performed for relief One month later roentgen examination showed that the sacculation had completely disappeared and that the contour of the esophagus was normal The gastric fistula was then permitted to heal Roentgen examination later showed the esophagus to be persistently normal

In Aurelius[18] case roentgen examination showing a nichelike area at the lower end of the esophagus was confirmed by esophagoscopic study At this examination a

small ulcer at the cardiac end of the esophagus was found. A biopsy was taken. Microscopic examination revealed glands resembling those of the stomach. An edge of the section resembled the appearance of a peptic ulcer.

Additional examples of the niche in the diagnosis of peptic ulcer of the esophagus appear in the contributions of Chaoul and Adam[19] and Friedenwald, Feldman and Zinn.[20]

2. In malignant infiltration of the esophagus there is apt to be a destruction of the normal mucosal markings. When the mucosal structure of the narrowed area of the esophagus in peptic ulcer is demonstrable it may exhibit no destruction of the normal mucosal folds.

3. In the case of ulceration of a new growth projecting into the mediastinal tissues, the area of ulceration will be of irregular configuration and will be associated



Fig. 60. (A, *Left*) Peptic ulcer of the esophagus (first oblique position). Note the narrowing in the distal portion of the esophagus with a niche. Note also the dilatation of the esophagus above the constricted region. (B, *Right*) Second oblique position. The niche is more prominent at this time.

Differential Diagnosis. Differential diagnosis of peptic ulcer of the esophagus from a new growth is based on the following:

1. In new growth of the esophagus there may be an area of irregular narrowing without any projection beyond the confines of the contour of the esophagus itself, or the lesion, if of a proliferating character, may produce irregular translucent areas by direct intraluminal invasion. In peptic ulcer of the esophagus the contour is smooth in character and the ulcerating lesion projects beyond the confines of the esophagus itself.

with other evidence of infiltration in that particular segment of the esophagus in which ulceration has arisen.

4. New growth of the esophagus is a progressive disease with gradual extension of the malignant process. In the case of a niche of a benign ulceration, roentgen evidence of the lesion may disappear after medical management.

5. Esophagoscopy and biopsy help definitely to differentiate the two disorders in the event of any doubt.

A peptic ulcer of the esophagus may also

be readily differentiated from a diverticulum The latter is more apt to occur on the anterior wall of the esophagus at its mid portion near the bifurcation of the trachea The diverticulum may be tent shaped or perfectly well rounded and is practically never associated with an area of stenosis At times the niche may appear to be separated from the rest of the esophagus as a result of inflammation edema and spasm This does not occur in the case of a diverticulum the neck of which as a rule is clearly demonstrable

In a borderline case the therapeutic test may be of considerable help in that while the niche of a peptic ulcer of the esophagus may disappear under medical management the diverticulum remains identical in size and shape under such treatment Finally, as has been said esophagoscopy will aid in differential diagnosis if any doubt still exists Such a possibility may arise in the case of the perforation of the diverticulum into the mediastinal tissues

Illustrative Cases The following case shows roentgenographic evidence of peptic ulcer of the esophagus

B R male aged 50 This patient gave a history of about 1 year of pain in the epigastrium and of difficulty in swallowing There were intermittent attacks of nausea and vomiting without blood He had lost 20 pounds since the onset He had a chronic cough and the sputum at times was blood tinged

Esophagoscopic examination revealed an area of constriction in the distal esophagus with a flat ulcer

Microscopic study of a specimen removed through the esophagoscope revealed a diffuse infiltration of lymphocytes and plasma cells

Roentgen examination (Figs 60A and B) revealed the presence of a smooth area of constriction in the distal portion of the esophagus Beyond this narrowed area was a niche typical of an ulcer in this region In view of the roentgenographic appearance the esophagoscopic confirmation the biopsy report of chronic inflammation and the fact that there was no evidence of malignancy this case may be considered as one of peptic ulcer of the esophagus

Following gastrostomy as well as dilatation of the strictured area there was a remarkable improvement in the condition as noted in the roentgen appearance (Fig 60C)

Figure 61 shows a niche at the distal end of the esophagus This region is narrowed but the mucosal structure is intact A diagnosis of peptic ulcer of the esophagus was made which was confirmed by esophagoscopy The narrowed area was successfully dilated and later esophagoscopy showed that the ulcer had disappeared About 2½ years later the patient was clinically well



FIG 60C Same patient as shown in Figure 60 (A) and (B) The appearance of the esophagus after dilatation of the stricture and gastrostomy

The association of an ulcer of the esophagus with herniation of the stomach is illustrated by the following case

S G female aged 52 The patient gave a 2 weeks history of severe right upper quadrant pain radiating to the back and not definitely related to food She vomited coffee ground material and had a tarry stool Physical examination of the abdomen was essentially negative She had marked secondary

anemia There was no evidence of weight loss In spite of transfusions, she had recurrent massive bleeding A left transthoracic resection was done for vagotomy There was complete fibrosis of the pleural cavity The proximal portion of the stomach was herniated through the esophageal hiatus An ulcer was found to have perforated into the lower end of the esophagus Closure of the perforation was unsuccessful because of the necrosis friability and extent of ulceration The ulcerated portion of the esophagus was resected an esophagogastrostomy was done

was shortened, I was not certain whether this was the result of a congenital anomaly or was secondary to the stricture causing shortening, with a consequent pull on the stomach drawing it up into the chest The surgeon thought the shortening was congenital Except for the bleeding which may have originated from the ulcer of the esophagus, there were no clinical symptoms to suggest a lesion in this region It is also noteworthy that the patient had a duodenal ulcer which might have explained the massive hemorrhage





Fig 61 (*Left*) Peptic ulcer of the esophagus

Fig 62 (*Right*) Peptic ulcer of the esophagus The arrow points to the niche in the narrowed segment of the esophagus at its junction with the herniated portion of the stomach

The surgeon described the esophagus as shortened probably congenitally

Pathologic diagnosis of the resected specimen was reported as follows extensive ulceration of the mucosa The ulcer base contains some fibrinoid material and connective tissue infiltrated by chronic inflammatory cells The fibrous tissue extends into the submucosa and the muscularis is partially interrupted and replaced by fibrous tissue The periesophageal tissue is thickened by connective tissue No evidence of tumor is seen Diagnosis Chronic ulcer of esophagus

Roentgen examination in addition to disclosing a duodenal ulcer showed some interesting features in the appearance of the esophagus (Fig 62) The distal end was narrowed at its junction with a supradiaphragmatic portion of the stomach At the strictured zone there was a cone shaped niche The diagnosis was peptic ulcer of the esophagus While it was obvious that the esophagus

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13

Esophagitis

ROENTGEN DIAGNOSIS

Zenker and Von Ziemssen gave one of the earliest descriptions of esophagitis. It is a rare affection.

The condition may result from mechanical, thermal or chemical irritation of the esophagus by food or foreign bodies. Inflammation may also result from external factors, such as conditions in the spine, the mediastinum and the lymph nodes. Also esophagitis may be a localized manifestation of a more general disease, as in some of the infectious diseases, acute exanthemata and possibly syphilis. The inflammation may be catarrhal with superficial erosions which frequently heal without leaving any scar. Catarrhal inflammation of long duration may lead to hypertrophic thickening of the mucosa, with the development of polypoid processes. This may in some cases also be associated with considerable hypertrophy of the muscular wall of the esophagus, especially when the condition is complicated by dilatation. Actual ulcers of considerable depth may develop; these may be capable of initiating hemorrhage through involvement of large blood vessels. Atony of the entire esophagus may result from a diminution of the contractile function of the esophagus, thus leading to diffuse ectasia.

The authors further described follicular, fibrinous, pustulous, phlegmonous and corrosive types of esophagitis. Ulcers of the esophagus may develop in all these types of inflammation.

Regurgitation of acid gastric secretion may be a factor in the development of some cases of esophagitis.

ROENTGEN DIAGNOSIS

In the deformity produced by esophagitis the area of involvement is apt to be narrowed. The contour is smooth. There is no sharp demarcation of the narrowed area of the esophagus from the rest of the smoothly outlined contour, the one passing imperceptibly into the other.

In the narrowing produced by a new growth, however, the area of involvement is irregular and there is sharp demarcation of this region from the rest of the esophagus. In esophagitis, while the mucosal appearance of the involved area is altered, with increased prominence of the folds and occasionally the presence of translucent areas because of superficial excrescences, these deformities, as a rule, never assume the marked irregularities produced by a new growth, invading the wall or extending into the lumen in polypoid fashion.

In a borderline case, esophagoscopy and biopsy may be essential in order to arrive at a differential diagnosis.

Illustrative Cases. The following reports illustrate cases of peptic esophagitis.

J. G., male, aged 58. For 8 months this patient noticed that meats, when improperly chewed, stuck in his throat before going down. He had no pain or evidence of bleeding. There was no increased difficulty in swallowing. Two years previously the patient had vomited blood and remained in the hospital for a week. He had lost about 12 pounds.

Esophagoscopy revealed a stricture of the esophagus, at the level shown in the roentgenogram, at the lower extremity of which there was ulceration of the mucosa. The presence of ulceration was noted on repeated examina

tions At no time did esophagoscopy reveal any evidence of new growth

A biopsy of tissue removed at the site of stricture was reported as 'chronic productive inflammation The patient gradually improved as a result of dilatation of the strictured area of the esophagus

Roentgen examination (Fig 63) revealed a very smoothly outlined area of stenosis at the junction of the lower and the middle thirds *of the esophagus with moderate dilatation* above this region Because of the smooth sharply outlined appearance of the area of constriction of the esophagus in the roentgenogram the strictured area was considered to be the result of a benign process In conjunction with the findings on esophagoscopic examination as well as the pathologic findings on biopsy *this may be considered a case* of peptic esophagitis

The next case has features of unusual interest

L W male aged 63 Six months before his admission to the hospital this patient complained of a sudden onset of vomiting On *three occasions he brought up blood He com*plained of difficulty in swallowing solid foods but he had no pain as a result of this difficulty in swallowing Gastrostomy was performed

The esophagoscopic examination revealed a firm fibrous cicatrizing stenosis 39 5 cm from the upper teeth This stenosed area was dilated through the esophagoscope

A specimen was removed for biopsy It proved to be scar tissue The patient s difficulty *in swallowing disappeared and the* gastrostomy opening was closed The stricture of the esophagus had apparently resulted from previous ulceration

Roentgen examination of the esophagus prior to esophageal dilatation (Fig 64A) showed an area of marked stenosis about 1 inch long Above it the esophagus showed considerable dilatation Following mechanical dilatation of the involved region the strictured area showed a considerable increase in lumen (Fig 64B)

This patient had been followed for a period of several years and there has been no return of the original stricture Repeated roentgenographic studies showed an appearance similar to that seen in Figure 64B In all probability, *this stenosis arose as the result* of ulceration with secondary constriction The history of the vomiting of blood at the onset of the disorder is clinically suggestive of active ulceration of the esophagus at that time

A well-controlled example of esophagitis is illustrated by the following case

F H male aged 58 During the preceding 3 years the patient had had increasing difficulty in swallowing food especially solids with a feeling that the food was sticking in his chest This was associated with substernal pain radiating to the back He vomited occasionally and bright blood was sometimes present He had lost 20 pounds in the preceding 3 months Physical examination re-



Fig 63 Smooth narrowing of the esophagus due to esophagitis

vealed a pale weak male There was tenderness in the epigastrium but examination of the abdomen was otherwise essentially negative

Esophagoscopy showed an ulceration of the mucosa just above the cardia A biopsy of this region was taken The pathologic report was acute and chronic esophagitis

A biopsy of the involved area of the esophagus was again taken 3 weeks later and pathologic examination at this time was reported as *hyperplasia of the esophageal* epithelium There was no tumor tissue A third biopsy 5 weeks later was reported as showing an acute inflammatory exudate



FIG 64 (A *Left*) Marked narrowing of the esophagus due to benign stenosis (B, *Right*) Same patient as is shown in (A) Note the marked improvement after dilatation of the stenosed area

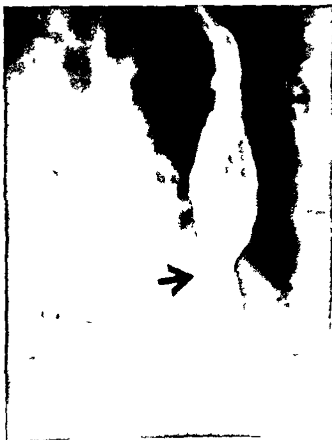


FIG 65 Peptic esophagitis Note the smooth narrowing of the esophagus

The patient was followed both in the out patient department and on a number of occasions on the ward Repeated esophagoscopic examinations were made, all of which were negative for new growth

Examination 3 years after the original esophagoscopy showed a smooth stricture at the distal end There was no evidence of new growth The stricture was dilated with considerable clinical improvement

Roentgen examination (Fig 65) showed a comparatively smooth narrowing at the junction of the lower and middle third interpreted as indicating a stricture of inflammatory origin which might be associated with ulceration even though a niche was not actually demonstrable The roentgenographic appearance represented the condition 3½ years after the original observation at which time a similar diagnosis had been made The diagnosis of benign stricture thus received further support from the duration of the deformity (3½ years) without any evidence that irregularity had developed in the narrowed zone The diagnosis was further substantiated by repeated esophagoscopic examinations and pathologic studies of biopsies taken on numerous occasions Also the patient benefited clinically from dilatation of the stricture under esophagoscopic control Incidentally a duodenal ulcer was found originally at the time when the esophageal stricture was first discovered The patient was later operated on for this and a partial gastrectomy was done The possibility arises that the duodenal ulcer may have antedated the esophageal lesion and that repeated vomiting of hyperacid fluid might have been an etiologic factor in the development of the esophageal inflammation

Because of recurrent episodes of dysphagia and hemorrhage operation was performed with resection of the distal 10 cm of the esophagus and an anastomosis of the remaining esophagus to the stomach The pathologic diagnosis of the resected segment was chronic esophagitis

The findings in the next case were confirmed by autopsy

J G male aged 59 gave a history of dysphagia of 2 months duration and a weight loss of 20 pounds He vomited undigested food which did not contain blood At first the dysphagia was limited to solid food but later he vomited liquids as well Ingestion of food was accompanied by pain which was relieved by vomiting Physical examination was essentially negative except for tenderness in the epigastrium

Autopsy revealed an intense esophagitis The inflammatory tissue consisted mainly of plasma cells and infiltrated the nerve ganglia and muscle fibers

Roentgen examination (Fig 66) revealed a smooth narrowing of the esophagus proximal to the cardiac end with secondary dilatation above The extreme distal portion of the esophagus was not involved thus differentiating it from the tapering of cardiospasm The smooth narrowing was characteristic of a benign inflammatory process as was verified at autopsy



Fig 66 Peptic esophagitis Note the smooth narrowing of the esophagus proximal to the cardiac end This differentiates the condition from cardiospasm in which the tapering occurs at the cardiac end

An important type of inflammatory pathology of the esophagus is that resulting from the swallowing of corrosives As a rule

the involvement of the esophagus is diffuse, with irregularity of contour and narrowing of the lumen In rare cases, however, the outline of the esophagus may remain surprisingly smooth but with evidence of constriction The region of involvement at times type of esophagitis or from the narrowing produced by an encircling mass such as a bronchogenic tumor may be difficult or impossible

When the localized area is of irregular outline and is associated with an abnormal





Fig 67 Marked stenosis of the esophagus due to lye
Fig 68 Diffuse irregular narrowing of the esophagus due to lye

may appear to be quite localized with narrowing and secondary dilatation smooth or irregular outline at the site of involvement, and sometimes considerable destruction of the mucosal folds

When the process is localized and the contour is smooth, differential diagnosis on the basis of roentgen evidence alone, from the benign constriction of a noncorrosive

mucosal relief differential diagnosis from an infiltrating new growth may also be extremely difficult and in some cases impossible on the basis of the radiologic evidence alone In these exceptional cases, however, the clinical history will offer the clue to the actual underlying factor involved in the production of the roentgen findings

The following is a case of this nature

FIG 69 Steno is of the esophagus due to lye
FIG 70 Stenosis of the esophagus due to lye

G P About 4 years before his admission to the hospital, this patient had swallowed an indeterminate quantity of lye A Levine tube was left *in situ* thereafter for 4 weeks The patient had frequent esophageal dilatations for 1½ years In the following year, he had no treatment because he was relatively symptom free Three weeks before admission to the hospital, his difficulty in swallowing returned and became progressively worse, until he could barely swallow water

The esophagoscope was passed and a stricture was noted with only a small pin point opening The stricture opening expanded and closed with inspiration and expiration Repeated dilatations of the esophagus were done The lumen gradually increased in size and the patient was discharged improved

Roentgen examination (Fig 67) revealed a marked stenosis of smooth outline involving the midportion of the esophagus with moderate dilatation of the esophagus above the area of constriction There was no evidence of any irregularity of contour anywhere throughout the esophagus The very sharply defined contour at the site of narrowing strongly favored the diagnosis of a benign constricting process The exact cause in this case could be determined only on the basis of the clinical history and the evidence on esophagoscopic examination



FIG 71 Stenosis of the esophagus due to lye with fistula

Figure 68 is an example of diffuse narrowing of almost the entire esophagus, with irregularity of contour, due to the swallowing of lye

Additional examples of stricture of the esophagus resulting from caustics may be noted in Figures 69 and 70

In the next case of stricture of the esophagus resulting from lye, a fistulous tract ultimately developed

M D female, aged 18 The patient had tried to commit suicide by swallowing a fifth of a glass of a combination of lysol and lye She vomited blood streaked material and was promptly removed to the hospital The following day she complained of a substernal burning pain and dysphagia A Levine tube was passed through the esophagus for feeding This was removed about 2 weeks later, following which she was able to eat the regular ward diet

Esophagoscopy at this time revealed a stricture without any hemorrhagic areas

After the patient was discharged from the hospital the stricture was treated by weekly dilatation of the esophagus In spite of this the dysphagia increased to such a degree that a Levine tube could not be passed A gastrostomy was done

Roentgen examination 9 months after the swallowing of lye (Fig 71) showed marked stricture of the esophagus with an irregularly outlined walled off fistulous tract originating at the junction of the upper and middle thirds of the esophagus What had evidently occurred was a perforation of the esophageal wall possibly induced at the time of one of the dilatations of the stricture through the esophagoscope

The patient continued to have increasing difficulty in swallowing as well as episodes of bleeding necessitating further surgical intervention At operation the esophagus below the arch of the aorta was found to be fibrous and scarred so that it was impossible to identify its muscle structure The involved portion of the esophagus was resected and an esophagogastrostomy was done

Pathologic examination revealed that a segment of the lower esophagus had been opened longitudinally The wall was definitely thickened throughout and the lumen appeared markedly narrowed at the lower end

Microscopic examination revealed (1) Chronic esophagitis (2) fibrosis of esophagus with hypertrophy of muscle (3) foreign body giant reaction

14

The Esophagus in Scleroderma

The first recorded cases of the association of dysphagia and scleroderma were those of Ehrmann,[1] Schmidt, Helm[3] and Schwarz[4] None of these authors, however had the opportunity of studying the underlying pathology of the esophagus responsible for the dysphagia although they suspected that the changes were part of the more generalized disease and similar to those in the skin

The proliferation of connective tissue in the wall of the esophagus to explain the derangement of function was described at autopsy by Matsui quoted by Kure[6] Hoesli[7] and Weissenbach[8] On histologic examination of the esophagus in their cases of scleroderma these investigators found induration of the submucosa degeneration of elastic tissue and hypertrophy of the muscularis mucosae as well as atrophy of the muscular layer with vacuolization and increased proliferation of connective tissue The presence of serious alterations in the structural integrity of the wall of the esophagus was further corroborated by the autopsy studies of Rake[9] Weiss[10] and Bevans[11] As a result of these changes in the wall of the esophagus there are a number of disturbances in its behavior which may be recognized on fluoroscopic and roentgen examination

1 There is a marked diminution or complete absence of peristaltic activity the lumen of the esophagus remaining wide open even though there is no stenosis

2 There is considerable delay in the emptying of the esophagus in the absence of constriction and the barium may cling to the wall for many hours Detailed studies of the disturbed function of the esophagus in scleroderma were recorded by Kure and his associates[6] in five cases

3 In some cases there may be an actual area of stenosis at the distal end of the esophagus Tessler and Pohl[1] in one of their roentgenograms showed an area of constriction about 2 centimeters in length and of smooth contour with secondary dilatation of the proximal portion This region was visualized by esophagoscopy but no tissue was removed for biopsy They considered the narrowing to be secondary to connective tissue proliferation with scarring Their radiographic findings were confirmed by other observers (Ochsner and DeBakey,[13] Jackson,[14] Weiss,[10] Lindsay, Templeton and Rothman,[1] Hale and Schatzki[16] and Olsen O Leary and Kirklin[17])

Lindsay and his associates[15] carried out biopsy studies of tissue removed through the esophagoscope from the area of stenosis, and microscopic examination showed changes in the connective tissue characteristic of the alterations produced by scleroderma in the skin As a result of the stricture the esophagus may be shortened and in the process may pull up the cardiac portion of the stomach into the chest through the hiatus (Olsen O Leary and Kirklin)[17] The appearance may mimic that of a congenitally short esophagus

4 In some cases disturbance in the behavior of the esophagus in scleroderma may be present in the absence of any subjective symptoms (Kure[6] Bevans[11])

Presumably therefore the organic involvement of the esophagus must be quite advanced before the patient complains of dysphagia

Illustrative Case Abnormalities of the esophagus in scleroderma are illustrated by the following case

M W female aged 53 During the preceding 4 years the patient noted that the skin over her hands became tense and appeared white over the knuckles The fingers became contracted with difficulty in extension which gradually became more marked During this period of 4 years the patient had difficulty in swallowing greater with solid food but also some with liquids The food seemed to stick substernally for about 2 hours Occasionally drinking water helped to push the food down Her dysphagia was lessened if she minced her food very fine and ate very slowly She had lost 20 pounds in the preceding 3 months

Physical examination revealed that some of the fingers were permanently flexed at the terminal phalanges A similar condition existed in the lower extremities and toes The skin over the hands was atrophic dry and tense The terminal phalanges of the second third fourth and fifth fingers of both hands were held in a flexed position increasing in flexion in that order There was some depigmentation of the skin over the heads of the metacarpals The nails showed changes with subungual keratosis of the index fingers There was some pitting and striation of the nails Similar changes were present in the toes There was wrinkling dryness and slight contraction of the skin about the lips



Fig 72 (A *Left*) The esophagus in scleroderma Very little of the barium entered the stomach during prolonged fluoroscopy in spite of repeated efforts at swallowing on the part of the patient No peristaltic activity was noted (B *Center*) Same patient as is shown in (A) Appearance one half hour after the swallowing of barium Note the marked delay in emptying (C *Right*) Same patient as is shown in (A) and (B) Constriction at the distal end of the esophagus seen in the second oblique position

Roentgen examination revealed hypertrophic osteoarthritis of both wrist joints There was considerable osteoporosis involving the bones of the hands and wrists There was absorption of the distal portion of the terminal phalanges of both thumbs more extensive on the left side but to some extent affecting the distal phalanges of the other fingers

Examination of the esophagus (Fig 72A) showed moderate dilatation throughout Very

14

The Esophagus in Scleroderma

The first recorded cases of the association of dysphagia and scleroderma were those of Ehrmann,[1] Schmidt-Helm[3] and Schwarz[4] None of these authors, however, had the opportunity of studying the underlying pathology of the esophagus responsible for the dysphagia although they suspected that the changes were part of the more generalized disease and similar to those in the skin

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Illustrative Case Abnormalities of the esophagus in scleroderma are illustrated by the following case

15

Syphilis and Tuberculosis of the Esophagus

SYPHILIS

Incidence Cornil[1] stated that syphilis rarely attacks the esophagus He quoted a case reported by Virchow in which a gumma was found at autopsy but he had encountered no such case in his own experience

Lublinski gave an excellent review of the older literature on syphilis of the esophagus and described two cases of his own these however were not corroborated by autopsy study The stricture in both these cases diminished following antisyphilitic treatment Both patients had given a history of syphilis and in one case the esophageal lesion was associated with a gumma of the tongue

Wilks[3] described a case of typical syphilis of the pharynx in which the ulcerations extended to the esophagus with resulting contraction of the esophagus

Wile[4] described a case of syphilis of the esophagus in a woman aged 40 with a history of dysphagia of about 9 years duration which had gradually increased in severity Esophagoscopy showed a marked sclerosis of the entire tube so that only the smallest sized filiform bougie could be passed through it into the stomach Roentgen examination showed two areas of stricture Excellent clinical response was obtained after antisyphilitic measures However dilatation of the stricture was also carried out with bougies of increasing size

Guyot collected 57 cases of syphilis of the esophagus reported in the literature to which he added 2 cases of his own Of these he stated that 11 cases were verified by examination at autopsy and 15 diagnosed esophagoscopically were cured by antisyphilitic measures He described three anatomic types (1) the presence of a gumma which may undergo ulceration (2) the spread of a syphilitic phlebitis or specific pneumonitis with extension to the esophagus or secondary perforation into it and (3) a diffuse chronic inflammation with generalized sclerosis and superficial ulceration All three forms may terminate in stenosis

Tertiary syphilis of the esophagus has been described by Wilcox[6] In a case which he examined roentgenologically an irregularity of contour involving almost the entire esophagus giving the appearance of a diffuse esophagitis was seen Improvement followed dilatation and antisyphilitic measures but no anatomic diagnosis was made

Because of the rarity of the condition possibly only those cases should be accepted which meet the criteria of anatomic and bacteriologic proof

Roentgen Characteristics Roentgenologically no specific characteristics have been noted and the appearance has simulated that either of a nonspecific inflammatory process or a new growth

TUBERCULOSIS OF THE ESOPHAGUS

Tuberculosis of the esophagus is extremely rare Rokitansky[7] in his famous work stated that tuberculosis of the esophagus is rarely if ever found and he warned that this must not be confused with tubercular degeneration of the neighbouring lymphatic glands He apparently had had no personal experi

little of the barium entered the stomach during prolonged fluoroscopy (about 15 minutes) *and in spite of repeated efforts at swallowing* on the part of the patient One half hour later (Fig 72B) a good deal of the substance was still clinging to the wall At this time there was a blunt abrupt termination of the distal end At the end of 13 hours, fluoroscopic observation still showed a small amount of barium in the esophagus The contour of the wall of the esophagus appeared quite smooth At times on examination in the second or left oblique position (Fig 72C), there appeared to be some constriction of the extreme distal end The walls of the esophagus at no time showed evidence of contraction but remained as if rigid Emptying appeared to occur primarily as the result of gravity and the occasional opening up of the distal cardiac end particularly under the influence of repeated acts of deglutition

An additional example of the roentgen appearance of the esophagus in scleroderma may be noted in Figure 326A, which is included later in this book in connection with changes which had occurred in the small intestine

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13 Carr D T and D M Spain Tuberculosis *in a carcinoma of the esophagus* Am Rev Tuberc 46 346 1942
14 Dean L W, and J B Gregg Report of a case of coexistent carcinoma tuberculosis and syphilis of the esophagus Ann of Otol Rhin & Laryng 26 619 1917

ence with a case of intrinsic tuberculosis of the esophagus

Flexner[8] described three types of tuberculous involvement of the esophagus In the first type, the esophagus is secondarily involved through contiguity, as when caseous bronchial nodes ulcerate through into the esophagus or when an abscess associated with caries of the vertebrae perforates into it Under this heading he also included secondary extension to the esophagus from tuberculous ulcers of the pharynx

In group 2, he included those cases in which the existence of a previous lesion predisposed the mucosa to tuberculous infection

To the third group belong those cases in which involvement of the esophagus occurred in the course of a general infection of the body in acute disseminated miliary tuberculosis

Flexner in his review of the subject of tuberculosis of the esophagus up to the time of the publication of his paper in 1893 stated that Spillman[9] in 1878 described the first case of an intrinsic lesion of tuberculous nature which had developed within the esophagus itself An ulcer was present 1 cm by 1 5 cm in the midportion of the esophagus with a barely perceptible perforation into the left bronchus The patient had a pleural tuberculosis

The only other cases that met the criteria (according to Flexner) of group 3 were those reported by Frerichs[10] in 1862 and Mazzotti[11] in 1885 To these reports Flexner in his article added a case from his personal experience

Denonvilliers[1] is frequently given credit for having reported in 1837 the first case of tuberculosis of the esophagus In his description, however it is to be noted that he considered the ulceration of the esophagus which had produced a fistulous communication with the trachea as being secondary to a tuberculous process in the intercellular space between the trachea and the esophagus This case therefore cannot be included in the third group described by Flexner

Although a number of additional cases of tuberculosis developing within the esophagus itself have been added in succeeding years, this type of involvement is still extremely rare and the esophagus enjoys great immunity to the direct implantation of the tubercle bacillus, particularly in the absence of any pre existing lesion of the esophageal mucosa

The rarity of tuberculosis of the esophagus is shown by the fact that, in 1,400 autopsies on tuberculous patients performed in the 10 years between 1932 and 1942 at Bellevue Hospital, there was only 1 case of tuberculosis of the esophagus In addition to this isolated case, Carr and Spain[13] described a case of a tuberculous lesion apparently engrafted upon a primary carcinoma of the esophagus

A somewhat similar case of tuberculosis developing within a carcinoma of the esophagus is described by Dean and Gregg[14]

There is no roentgen alteration in the appearance of the esophagus specifically ascribable to an intrinsic tuberculous lesion

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outstanding factors These are enumerated below

1 The appearance of the stomach in the chest cavity above the diaphragm

2 The delineation of the definitely shortened esophagus joining the stomach within the thorax

3 The demonstration of definite rugal folds within the 'thoracic stomach'

with it into the chest cavity The lower portion of the esophagus will then appear kinked on itself and somewhat tortuous One will obtain the impression that if the hernia could be reduced the entire esophagus would straighten out and would appear normal in length making its exit below the diaphragm

Clinically such differential diagnosis is

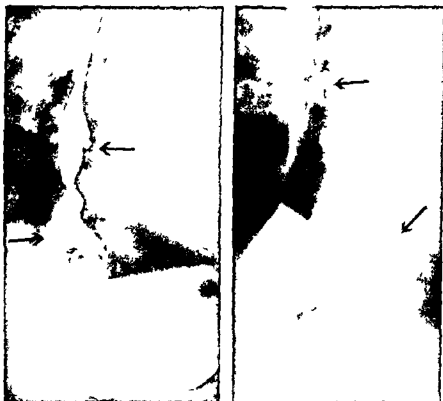


Fig 73 (A *Left*) Thoracic stomach with diverticulum of the esophagus (B *Right*) Same patient as is shown in (A) Detailed appearance of the diverticulum of the short esophagus and the thoracic stomach

In the differential diagnosis the possibility of a true herniation of the stomach must be considered The roentgen findings in gastric herniation will be discussed in greater detail later Suffice it to say here that in this condition the esophagus retains its normal length However, one must carefully guard against the fact that in true herniation of the stomach the lower portion of the esophagus may be carried up

of great importance from the standpoint of possible surgical intervention While a true hernia of the stomach may be reducible obviously the congenital short esophagus type of abnormality does not lend itself to such treatment

Another possibility is that of an ampullary dilatation at the cardiac end of the esophagus This may arise as a result of temporary failure of relaxation at the

16

"Thoracic Stomach" (Congenitally Short Esophagus)

ROENTGEN DIAGNOSIS

'Thoracic stomach' is a rare anomaly in which part of the stomach is in the chest cavity not as the result of actual herniation but in association with a congenitally short esophagus

Embryologically, according to Keith [1] the stomach originates *in the thorax* It *then* migrates into the abdomen Following this there is a fusion of the diaphragm except for the esophageal hiatus Should the stomach fail to descend normally, two results may follow (1) the esophagus may remain permanently shortened a part or all the *stomach remaining in the thorax* or (2) the fusion of the diaphragm may occur before the stomach has made its complete descent into the abdomen o that the opening is much larger Finally the stomach completes its migration and the large hiatus remains

The 'thoracic stomach occurs when the *esophagus remains permanently shortened* The condition, therefore is not a herniation in the true sense of the word In true herniation as will be discussed more fully later the esophagus is of normal length and is to be traced through the diaphragm The herniated portion of the stomach may vary *in position at times it may be present in* the thorax and at other times it may descend back into the abdomen This never can occur with the "thoracic stomach since its position in the chest is fixed by the short, unyielding esophagus Other viscera besides the stomach may be present in the thorax *in true diaphragmatic herniation*

The first case of a congenitally short esophagus was described by Richard Bright in 1836 The patient was a girl, aged 19, whose autopsy revealed the presence of the stomach encroaching upon the thoracic cavity

The name "thoracic stomach' was given to this condition by Percival Bailey [3] In his case he showed at autopsy that the body *of the stomach was present behind the pericardium* in the posterior mediastinum It was covered by a serous sac continuous with the peritoneum through the opening of the diaphragm The esophagus ended at the level of the third costal cartilage There was, therefore, no evidence of herniation of *the stomach into the thorax The condition* was due to the stomach having developed within the thorax itself

Of considerable value in the demonstration of the exact nature of 'thoracic stomach are the findings obtained on esophagoscopic examination Thus Findlay and Kelly [4] *demonstrated by esophagoscopy and* direct inspection beyond the area of stenosis as well as by microscopic study of mucosa removed from this region that the partial thoracic stomach was actually gastric in nature Fluid removed from this region was invariably acid to litmus [5]

Esophagoscopic examination in some of these cases has also shown that, at the region of communication between the short esophagus and the thoracic stomach evidence of chronic esophagitis and peptic ulceration may not infrequently be found

ROENTGEN DIAGNOSIS

The roentgen diagnosis of this condition depends on the demonstration of three

lationship is purely coincidental for the following reasons (1) diverticula of the esophagus are comparatively common although thoracic stomach is rare (2) the location of the diverticulum on the anterior wall near the bifurcation of the trachea is the common site of diverticula of the traction variety and (3) the pulsion origin of the diverticulum as the result of obstruction at the distal end of the esophagus, is not definitely tenable since in those cases of considerably more marked obstruction of the esophagus as in cardiospasm the presence of a diverticulum of the esophagus is rare

Another example of thoracic stomach is as follows

M B female aged 47 The chief complaint of this patient was difficulty in swallowing for about 1 year In addition the patient complained of substernal pain aggravated by food She was able to swallow liquid and soft foods easily but not bulky foods These seemed to stick substernally

Roentgen examination revealed a short esophagus associated with a thoracic stomach (Fig 74A) There was narrowing of the esophagus at its junction with the stomach Figure 74B shows the thoracic stomach with the characteristic rugal structure

The patient s symptoms became progressively worse Pain and burning appeared in the epigastrium promptly after the ingestion of solids or liquids Two years after the original roentgenograms she was referred to the hospital from the out patient clinic She had lost 30 pounds in the preceding year and vomited frequently Esophagoscopy was reported as revealing an annular mass with definite ulceration at the cardiac end While on the ward she developed a spreading infection from an acute parotitis

At autopsy in addition to a bilateral suppurative parotitis and coronary atherosclerosis there was a stricture of the esophagus

The microscopic examination revealed the following findings Section shows a greatly thickened wall and a poorly preserved mucosa which is absent completely except for one small cluster of stratified squamous epithelial cells The latter appears normal and is confined to the basement membrane In the central portion of the section it is thickest and here there is almost no recognizable remaining muscle tissue The wall is composed of fibrous tissue which in most areas is very dense with hyalinized collagen fibers while in other areas there is a more cellular connective tissue with fibroblasts and occasional clusters of lymphocytes and plasma cells The blood



Fig 74 C Same patient as is shown in Figure 74 (A) and (B) Shortly before death showing marked constriction at the junction of the esophagus and the thoracic stomach

vessels are congested in certain areas and in others are obliterated by fibrous tissue There are no epithelial structures in the wall In adjacent areas of the esophagus there are scattered areas of fibrosis and the muscle wall appears to be hypertrophied in one area

The diagnosis was Chronic inflammatory tissue with marked fibrosis

Roentgenographic examination shortly before death (Fig 74C) showed a marked increase in the degree of constriction of the

cardia The feaures that will aid in differential diagnosis are (1) the inconstancy of this finding during the course of a single examination or on repeated examination (2) a study of the mucosal relief will fail to show the presence of the rugal structure characteristic of the stomach

A diverticulum involving the distal area of the esophagus will on careful examination, show the presence of a neck communicating with the esophagus above the extreme cardiac end which will be traced entering directly into the stomach Moreover mucosal folds are never found within the confines of a diverticular sac

Illustrative Cases The following cases are illustrative of thoracic stomach or so called herniation of the stomach of the short esophagus type

E L, female, aged 60 This patient gave a history of difficulty in swallowing associated with substernal pain of 6 years duration At times, a piece of food appeared to get stuck substernally, following which she would be unable to eat for a few minutes Occasionally, she regurgitated solid food and blood streaked material During these years there was no exaggeration of the symptoms There were fluctuations in weight, so that weight lost at one time was recovered later on

Roentgen examination (Fig 73A and B) showed a short esophagus narrowed at its junction with a 'thoracic stomach" The esophagus showed a diverticulum originating from the anterior wall of its midportion



Fig 74 (A *Left*) Thoracic stomach with a short esophagus (B, *Right*) Same patient as is shown in (A) Note the rugal folds within the thoracic stomach

This association of 'thoracic stomach and esophageal diverticulum brings up three possible explanations (1) since thoracic stomach is a congenital anomaly that the diverticulum of the esophagus is also of congenital origin in this case (2) that the diverticulum of the esophagus is pulsion in origin as the result of obstruction at the distal end of the esophagus over a period of many years or (3) that the association is entirely coincidental in nature

I am inclined to the opinion that the re

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esophagus at its junction with the stomach The contour however, was quite smooth and faded gradually into the normal wall The pre autopsy roentgen diagnosis was benign stricture which had become more accentuated since the original examination 2 years before There was no evidence of a niche in the roentgenogram of the esophagus In the autopsy specimen however there was definite evidence of an ulcer proximal to the zone of constriction This region was apparently obscured by the barium above the constriction

Figure 74D shows the appearance of the esophagus *as noted at autopsy* with an ulcer proximal to the strictured area



FIG 74 D Same patient as is shown in (C) Appearance of the esophagus at autopsy with an ulcer proximal to the strictured area

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THE STOMACH

17

The Normal Stomach

TECHNIC OF ROENTGEN EXAMINATION

Before beginning fluoroscopic examination of the alimentary tract the heart and lungs are scanned in routine fashion. Even though the primary purpose of the examination is to determine the presence of any abnormality of the alimentary tract, such visualization of the chest may offer information of considerable value. In the first place, lesions of the heart and lungs of a coincidental nature may be found. Secondly, some of the pathologic findings discovered in this manner may throw considerable light upon the abdominal symptoms for the investigation of the cause of which the examination was primarily undertaken.

Thus an unsuspected lesion of the lung of a tuberculous nature may account for the patient's dyspepsia and loss of weight. An aneurysm of the aorta or an enlarged heart may explain distress vaguely localized to the upper abdomen. Moreover, direct evidence of the presence of a viscus within the chest may also be discovered, particularly the presence of herniation.

The freedom of mobility of both diaphragms should be determined as well as their relative position. Abnormalities of diaphragmatic behavior may be discovered which may be responsible for the abdominal symptoms. Marked elevation of the diaphragm may be due to atrophy of the diaphragm on the left side.

On completing this routine inspection of the chest, the abdomen itself should also be studied with considerable care. Opaque shadows due to urinary calculi may be noted and occasionally even calcified processes in the gallbladder, liver or spleen may be observed. In addition, various other evidences of calcification may be present due to lymph nodes, phleboliths and cysts. The amount of gas throughout the alimentary tract can be observed and in exceptional cases the diagnosis of intestinal obstruction may thereby be made in an individual in whom this condition had not been suspected clinically.

Direct visualization of the air bubble of the stomach may yield important findings. Instead of the normal contour, it may be deformed by encroaching shadows due to new growths.

After this superficial investigation as just outlined, one is prepared to continue with the more detailed investigation of the stomach itself.

The barium suspension is then administered. Under ordinary circumstances, barium sulfate is best administered suspended in water. The patient is given a glassful of equal parts of barium and water thoroughly stirred just before ingestion. A suspension of this kind will enter small areas much more readily than if the barium

of the stomach under living conditions in both health and disease

Not without opposition however did this new conception gain recognition Stiller believed that the appearance of the stomach as visualized roentgenologically following the ingestion of bismuth did not represent the true anatomic configuration of the stomach but was the result rather of the specific effect produced by the bismuth itself Hesse[1] however used a meal made with ground bone which because of its calcium was opaque to the roentgen rays and he demonstrated an identical delineation of the stomach He therefore showed that the configuration of the stomach as visualized roentgenologically was not peculiar to the ingestion of the bismuth salt

Also to disprove the contention of Stiller, Groedel and Seyberth performed the following experiment They worked with dogs and in order to make the stomach of the dog visible roentgenologically they sutured 30 perforated silver pearls along the greater and lesser curvatures The animal was then permitted to recover completely After administering bismuth or barium they found that the changes in the shape and position of the stomach were identical with those produced by the ingestion of a similar quantity of meat

Roentgenologically the stomach is divided into several regions which of course fade imperceptibly into one another The proximal portion containing the air bubble is referred to as the pars cardia That part of the stomach extending from the incisura angularis to the pyloric ring is referred to as the pars pylorica The portion of the stomach between the pars cardia and the pars pylorica is described as the pars media Extending from the pars pylorica to join the base of the duodenal bulb is the short stemmed pyloric ring

Rieder[3] considered the normal stomach to be shaped like a fishhook Holzknecht[4] believed the steer horn type of stomach to be the normal one Soon however increasing experience showed that both the Rieder and the Holzknecht types of stomach were within the range of the normal Moreover it was found that between these two distinct types there were all sorts of gradations the type of stomach demonstrated varying with the architectural structure of the particular patient under investigation

Other factors influence the position of the stomach Among the extrinsic causes are the state of contraction of the abdominal wall and the position of the small and large intestine Particularly important in this connection is the effect of the colon Considerable gaseous distention may cause almost every degree of displacement of the stomach and may occasionally be of such bizarre nature as to simulate changes of contour produced by organic disease Also other viscera may displace it In addition to the significance of these extrinsic factors Forssell[5] showed that the musculature of the stomach itself has considerable influence in causing variations in the position and contour of the stomach a mechanism which enables the stomach to accommodate itself to the conditions of its environment Variations in the position of the patient obviously also influence the position of the stomach

The size the shape and the position of the stomach therefore show considerable alteration even under normal conditions because of the following three reasons

1 The amount of food or other material within the stomach will produce variations which will modify its contour and anatomic relations A stomach apparently large in size when distended will appear much smaller later on in the process of digestion or after peristaltic evacuation of its contents

2 The position and the shape of the stomach will be subject to considerable alteration because of the nature of those structures within the abdomen that are in environmental relationship to it such as the size and degree of the gaseous distention of neighboring viscera

3 The nature of the intrinsic muscula

were mixed with food or made too thick in any other way Moreover, this thin fluid medium leaves the stomach more readily by way of the pylorus, and the prompt visualization of the duodenum and small intestine is thereby aided For special studies there may be modifications in the preparation of the barium suspension

The examination is best conducted in the morning, the patient having been instructed to abstain from food and drink since the night before This is important, as these substances particularly food will obscure the image and make detailed study of the stomach impossible

The patient, placed behind the fluoroscopic screen and facing the examiner, is *then turned obliquely* so that the right side is against the screen As described in the study of the esophagus, this is referred to as the 'first oblique position' The posterior mediastinum is thereby brought into clear relief The patient is then instructed to drink the barium suspension, and, under fluoroscopic control the passage of the opaque substance is observed as, one mouthful at a time it delineates the esophagus and is soon seen making its exit through the cardiac end as it enters the stomach itself In addition observations are made as the patient is turned through every degree of obliquity This includes what is spoken of as the second oblique position during which the left side of the patient is placed against the fluorescent screen Examination in the prone, and occasionally even in the Trendelenburg position may be necessary

As the first mouthful of barium enters the stomach, it is spread manually over the entire stomach with compression so as to determine the roentgen appearance of the structure of the gastric rugae and any evidence of abnormality as produced by intrinsic disease The view that there is a greater tendency for fluids to run along the lesser curvature the so-called Magenstrasse, is not substantiated by fluoroscopic observation As one observes the ingested barium entering the stomach it frequently flows downward in almost every direction

After visualizing the mucosal folds, the stomach is then completely filled with barium and an inspection of the contour is made Here too both fluoroscopic and radiographic observation through every degree of obliquity, both in the erect and in the prone positions, may be essential for complete scrutiny of any possible organic derangement

In addition, observation is made regarding the size and functional behavior of the stomach as it is influenced by tone and peristaltic action Roentgenograms may be taken without direct fluoroscopic control, or 'spot" films may be made of the exact area under suspicion Various methods of obtaining 'compression of localized areas have been developed, with instantaneous radiographic exposure after an ideal field has been brought into focus under fluoroscopic control

Films also may be taken of the stomach in the prone position as it gradually empties itself with exposures every 10 or 15 minutes By developing these films promptly and by making additional exposures, if indicated, it may be possible to obtain excellent roentgenograms depicting the mucosal structure of the entire stomach without the danger of distortion which may result from exaggerated mechanical compression

The exact technic used, however, and the number of exposures that are made, as well as their frequency will depend on the nature of the particular problem involved

ROENTGEN CHARACTERISTICS OF THE NORMAL STOMACH

With the advent of the roentgen method new light was thrown upon the normal anatomy of the stomach and ideas regarding its form and position as obtained from post mortem anatomy had to be completely altered Here indeed was an opportunity of studying both the form and the function

lesser and the greater curvatures at the cardiac end and by comparing it with the distance between two similar points in the pyloric region In the case of normal tonicity the distance measured at the cardia and at the pylorus is essentially the same In hypertonicity the distance between the two curvatures gradually diminishes from the cardia to the pylorus

Hypertonicity may not only manifest itself by a change in the shape of the stomach but may also be demonstrable through alteration in the roentgen appearance of the gastric mucosa Through the activity of the muscularis mucosae there may be an increased prominence of the rugal folds This effect upon the relief picture may show itself in two ways (1) by an increased toothlike irregularity of the greater curvature, and (2) by an exaggeration of the folds throughout the rest of the stomach This appearance may closely simulate that seen in the presence of organic disease

Atony In gastric atony if the distance between the lesser and the greater curvatures beginning at the cardia is traced distally it is found to increase so that by the time the pylorus is reached this distance between the curvatures shows a considerable exaggeration As barium enters an atonic stomach it sinks to the lower pole where it spreads out along the greater curvature without being maintained in a vertical column reaching to the cardia as is noted in the case of normal or increased gastric tone With diminished tone the lower pole of the stomach sags in a baglike manner Owing to the poor tone the pyloric portion of the stomach is visualized with considerable difficulty Either the ingestion of a considerable amount of barium may be essential or upward displacement of its contents by manual palpation may be required Compression of the barium towards the pyloric ring may also be necessary in order to force it into the duodenum so that that region may be visualized The emptying time of the stomach may not infrequently be prolonged beyond the 6 hour limit for normal function



Fig 75 Transverse hypertonic type of stomach in a hypersthenic individual

The elongated baglike appearance of the stomach is however, readily differentiated from gastric enlargement due to pyloric stenosis by the following characteristics

ture will also modify, to a large degree the apparent size, position and contour of the stomach

In addition to the visualization of the form of the stomach the roentgen method makes possible a detailed study of its functional behavior Peristalsis may be noted to begin ordinarily in the proximal portion of the stomach as superficial waves, which become deeper and more marked as they gradually approach the pylorus, particularly distal to the incisura angularis Peristaltic activity is usually not so prominent along the lesser curvature

The original conception was that the pyloric antrum was a definite structure anatomically preformed and possessing a sphincter antri Kastle, Rieder and Rosenthal[6] devised a bioroentgenograph for making a large number of exposures during a single respiratory pause each exposure being reduced to a small fraction of a second This enabled them to study the course of peristaltic activity without the aid of fluoroscopic observation Through the application of roentgen cinematography they showed that the antrum of the stomach varied from moment to moment This constant alteration of contour proved that the antrum was the result of gastric function and not of rigid anatomic formation These peristaltic waves were found by Kaufmann and Kienbock[7] to have an average duration of about 21 seconds There was no evidence of any relationship of peristalsis to either gastric acidity or cardiac activity

THE STOMACH IN INFANCY

Lewis[8] examined five stomachs from embryos ranging between 10 mm and 45 mm in length In the youngest embryo (10 mm) the stomach is already divided into an expanded pars cardiaca and a tubular pars pylorica Between these two areas is the incisura angularis In the 16 mm embryo the body of the stomach may be recognized along the lesser curvature, separating the esophageal cone from the incisura angularis By the second month of fetal life the fundus may be well developed

There is no definite normal type of stomach in the newborn, since this organ may show considerable variation in contour in different infants and in the same infant at different times[9]

These findings were corroborated by Bouslog and his associates[10] in the roentgenographic examination of 133 infants varying in age from 1 week to 6 months There was considerable variation in both the size and the shape of the stomach within normal limits Moreover barium retention in the stomach after 8 hours was occasionally noted in the apparently normal infant s stomach

THE TONUS OF THE STOMACH

Tone is that power of the stomach which enables its wall to enfold the gastric contents and maintain it in a more or less uniform column Tone, therefore, has a definite influence upon the form of the stomach In fact, Schlesinger[11] originally believed that differences in form depended entirely upon concomitant variations in tone He distinguished four types of stomach (1) hypertonic (2) orthotonic, (3) hypotonic and (4) atonic

That tone plays an important role in affecting gastric form was demonstrated experimentally by the work of Klee[1] with decerebrated cats Under these conditions the stimulating effect of the vagus and sympathetic nerves is greatly enhanced Increased stimulation of the vagus nerve caused contraction of the stomach and active peristalsis with increased emptying of the stomach and concomitant variations in shape

Gastric tone is therefore dependent upon two factors (1) the peculiar structural anatomy of the stomach and (2) the state of vagus and sympathetic activity

Hypertonicity Hypertonicity has a definite effect in altering the shape of the stomach Increase of tone may be determined by judging the distance between the

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The elongated baglike appearance of the stomach is however readily differentiated from gastric enlargement due to pyloric stenosis by the following characteristics

1 The gastric enlargement is downward toward the pelvis in atony, and practically never does it extend considerably to the right of the midline as in organic pyloric stenosis

2 The contour of the pyloroduodenal region *may be shown to be perfectly normal* in the case of functional atony, whereas in pyloric stenosis irregularity or abnormality *of contour in this region may be demonstrable*

3 While there is a delay in the emptying of the atonic stomach it never reaches the degree of gastric retention which is seen *as the result of organic obstruction* *In the* event of a 24 hour gastric retention one may be certain that the cause is organic

R W Mills[13] in 1917 showed the relationship of the variations in form tone and position of *the stomach to the general archi*tecture of the body He divided the types as follows first the hypersthenic, corresponding to the hypertonic type of Schlesinger or steer horn type of Holzknecht In this type *the gastric motility is fastest* and the tonus most marked. Secondly, at the other end of the scale, is the asthenic, corresponding to the atonic type of Schlesinger or the "congenital enteroptotic" type of *Stiller* *Between* these two extreme types are various gradations

A word of caution is essential regarding *the determination of the position of the* stomach This should be done with the patient erect since a stomach which hangs low may appear transverse when the patient is prone A stomach of normal or even *diminished tone may* thereby *mimic* the appearance of a hypertonic stomach merely by change of position from the erect to the prone The presence of an air bubble in the pars cardia with a fluid level will orient *the observer as to the erect position of the* patient at the time of the examination



Fig 76 Stomach exhibiting normal tonus (in a sthenic individual)

Illustrative Cases Figure 75 is an example of a hypertonic type of stomach

Note the gradual decrease in the distance between the lesser and the greater curvature of the stomach from the pars cardia to the pars pylorica (hypersthenic individual)

Figure 76 is an example of a stomach pars cardia towards the pars pylorica (hyposthenic individual)

Figure 78 is an example of an atonic type of stomach Note the baglike appearance of the lower portion of the stomach (asthenic individual)



Fig 77 Hypotonic stomach in a hyposthenic individual

exhibiting normal tonus (sthenic individual)

Figure 77 is an example of a hypotonic type of stomach Note the gradual increase in the distance between the lesser and the greater curvatures of the stomach from the

Figure 79 illustrates hypermobility of the stomach so that in the prone position most of the proximal portion of the stomach appears well below the duodenal bulb

Figure 80 illustrates volvulus of the stomach with rotation to the right This appear

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Illustrative Cases Figure 75 is an example of a hypertonic type of stomach

an abnormal position of the stomach is due to cascading and is associated with gaseous distention of the colon one may prove this point in two ways first, by administering additional quantities of barium to overcome the pressure exerted by the colon and noting the fact that the stomach assumes a normal position secondly, by the preliminary administration of castor oil the night before examination to diminish the amount of gas present in the colon Or we may combine both these procedures in a restudy I have not uncommonly seen strange abnormalities in the position of the stomach yield to the application of these procedures with a return to the normal thereby disclosing the nature of the mechanism involved

DISPLACEMENT OF THE NORMAL STOMACH BY EXTRAGASTRIC TUMORS

Extragastric tumors may displace the stomach in almost any direction The relation of tumors of pancreatic and of splenic origin to the position of the stomach will be discussed in later chapters dealing with lesions of the pancreas and the spleen

Illustrative Cases A retroperitoneal tumor may displace the stomach to the right

M S female aged 9 The child gave a 3 year history of recurring attacks of epigastric pain which was continuous for 24 hours There was occasional vomiting with the attacks She weighed 50 pounds Physical examination revealed a large mass in the left upper quadrant Operation disclosed a tumor apparently originating from the left adrenal gland Complete removal could not be accomplished

Pathologic examination of biopsy medullary carcinoma of the adrenal gland

Roentgen examination (Fig 83) showed a large mass displacing the stomach to the right and the loops of small intestine in a downward direction There was no evidence of any intrinsic involvement of the stomach The mass was obviously of extragastric origin As noted above the tumor was a malignant lesion of the adrenal gland

An unusual cause of upward displacement of the stomach is a cyst of the omentum

Gairdner[14] in 1851 gave the first description of a cyst of the omentum The cyst was found in a woman at autopsy, beneath the anterior layer of the greater omentum and consisted of a highly transparent closed sac between 3 and 4 feet in length and from ½ to 1½ inches in breadth It was lobulated in appearance The fluid in the sac was a



Fig 80 Volvulus of the stomach with permanent displacement to the right

transparent colorless serum No ova or parasites were present He considered it as very probably a variety of simple serous cyst

The next recorded case is by Bantock[15] in 1881 This was a large thick walled cyst which was present in the great omentum of a woman aged 58 A drawing accompanies the presentation showing the cyst enclosed by the layers of the omentum The case was presented by his associate Dr Doran who had assisted at the removal of the cyst In addition, he illustrated this condition by exhibiting a Hunterian speci

ance was persistently present throughout prolonged *fluoroscopic examination* as well as at repeated observations at later periods

An important alteration in the configuration of the stomach is that known as the cascade stomach In many cases this is *apparently associated with the pressure of* a gas distended colon The mechanism involved is discussed under functional disorders of the colon A marked example of a

1 By visual demonstration of the association *of the two conditions*

2 By the disappearance of the cascade deformity if the stomach is overdistended with barium, so as to overcome the pressure of the distended colon

3 By the disappearance of the cascade *deformity if the colon is emptied of its gas* after the administration of a cathartic or after an enema

4 By the fact that by artificially dis-





Fig 78 Atonic stomach in an asthenic individual

Fig 79 Hypermobility of the stomach with upward displacement in the prone position

typical cascade stomach is shown in figure 81 Note the cup and spill type of stomach, with the pyloric arm and the duodenal bulb directed downward and to the right Note also the enormous gaseous distention of the colon in direct proximity to the stomach The patient J A aged 46 gave a history *of marked constipation for 2 years and* occasional pain after eating After taking a laxative he obtained relief

That the typical cascade stomach is the result of pressure of a distended colon is shown in a number of ways

tending the colon with air, a cascade deformity of the stomach can be produced

A bizarre type of cascade stomach is shown in Figure 82A The pars cardia is actually at a lower level than the pyloroduodenal region There was considerable gaseous distention of the splenic flexure That this appearance was of a transient nature and not due to any intrinsic organic disease is shown by the fact that on reexamination 1 week later, the anatomic position of the stomach is essentially normal (Fig 82B) When it is suspected that

the inferior border of the stomach and was situated near the transverse colon The cyst was excised

The pathologic diagnosis was lymphangioma with cyst formation [1]

Roentgen examination (Fig 84A) showed upward displacement of the stomach with a smooth pressure defect indicating the presence of an extragastric mass as the cause The tumor was believed to be a cyst either of mesenteric or of omental origin The appearance of the cyst is noted in Figure 84B

lined concave depressions The eleventh and twelfth dorsal and the first and second lumbar vertebrae are the ones most commonly involved in the erosive process (4) when a globular mass in anatomic relationship to the abdominal aorta shows a combination of calcification of the rim and vertebral erosion, the diagnosis of aortic aneurysm may be made with assurance

Kampmeier [1] noted that evidence of



Fig 82 A Note this bizarre type of cascade stomach

pressure erosion of the vertebrae was the most important finding in aneurysm of the abdominal aorta Of 32 cases radiographed a roentgen study of the spine was made in 24 and in 18 of these (75 per cent) erosion of the vertebrae was present Four of this group showed gastric displacement by the aneurysm as well as erosion and in 1 of these 4 cases a gastric deformity was produced by the pulsating mass The most commonly eroded vertebrae were the twelfth dorsal and the first lumbar

Another clinically important cause of displacement of the stomach is aneurysm of the abdominal aorta Aid in the roentgen recognition of this condition may be obtained from (1) evidence of partial calcification in the wall of the mass which as a rule is globular in appearance (2) the close anatomic relationship of this mass to the position of the abdominal aorta and (3) evidence of erosion of the vertebrae due to pressure of the aneurysm Because of the fibrocartilaginous nature of the intervertebral disks they resist destruction and the pressure of the aneurysm is exerted primarily on the anterior portion of the bodies of the vertebrae producing irregularly out

The frequency of erosion of the vertebrae in abdominal aneurysm was also shown by Hubeny and Pollack, Eliason and McNamee [3] Scott [24] and Pratt Thomas [*]

men of a small cyst entirely within the folds of the great omentum

From then on there were reports of isolated cases so that Hasbrouck[16] in 1908 was able to review 34 cases of which he considered only 24 as acceptable Twelve of these originated from the omentum by a pedicle and were not true cysts within the layers of the omentum He added a case of his own a cyst originating from an endothelioma of the omentum

Additional cases were reported by Dowd[17] in 1911 and Pybus[18] in 1917 Pybus case was in a child of 4 The photograph of the patient shows a hugely distended abdomen At operation a thin walled cyst was situated in the great omentum At its upper pole it had to be peeled from the greater curvature of the stomach in order to be freed

By 1935 Horgan[19] had found 97 acceptable cases in the literature The condition is apparently more common in children 35 per cent having occurred in the first decade and 68 per cent during the first 30 years Horgan's case of omental cyst occurred in a child 12 years old

There is a paucity of descriptions of the radiologic findings in this condition In Birnkrant s case radiographic examination showed marked downward displacement of the transverse colon [9]

In the following case, the stomach was displaced upward by an omental cyst

M S, female, aged 2½ years During the preceding 3 months the child had passed bright red blood by rectum on five or six occasions this was accompanied by diarrhea One month later she complained of pain in *the back which gradually became worse* Two days before admission to the hospital the pain in the child s back was accompanied by persistent vomiting fecal in odor The next day the pain shifted to the abdomen

Physical examination revealed a mass which felt cystic just to the left of the mid line

At operation there was a large cyst in the greater omentum the size of a grapefruit There was an extension of the mass, which contained numerous small grapelike cysts The omental cyst created a pressure defect on



FIG 81 Cascade stomach Note the marked gaseous distention of the splenic flexure

connected to the eleventh and twelfth thoracic and the first and second lumbar vertebrae which have been irregularly eroded to a maximum of 2 cm The sac is filled with clotted blood The surface is flexible but here and there contains calcified atheromatous plaques On the right anterior surface there is a defect leading into the right pleural cavity which is filled with clotted blood The esophagus and stomach are normal

well rounded saccular area There was an irregular distribution of calcification in the rim of this mass Note also the erosions of the vertebrae produced by the pressure of the aneurysm

The diagnosis of an abdominal aneurysm as the cause of the displacement of the stomach not only depended on its location and its rounded contour and calcified rim



FIG 83 Displacement of the stomach by a carcinoma of the adrenal gland

but received further support from the fact that the lower dorsal and upper lumbar vertebrae were eroded

The retrogastric pressure of an abdominal aneurysm may distort the appearance of the stomach in such a way as to simulate the deformity of an intrinsic malignant lesion

The final diagnosis was syphilitic aneurysm of the aorta with rupture of the aneurysm and hemorrhage into the right pleural space

Roentgen examination of the stomach (Fig 86A) revealed evidence of considerable pressure upon the lesser curvature of the proximal portion of the stomach with considerable displacement to the left The stomach itself showed no evidence of any intrinsic organic disease The cau e of the displacement was best seen in the lateral film (Fig 86B) which showed a fairly

T M male aged 63 The patient complained of substernal pain and at the time of admission to the ho pital was suffering from a severe psychosis with hallucinations and

Displacement of the stomach by an abdominal aneurysm may be noted in the following examples

An aneurysm of the abdominal aorta may displace the stomach to the left The patient, J N, aged 44, was hospitalized because of intermittent upper abdominal pain of brief duration and a small pulsating abdominal mass, which he had noted for several years Physical examination revealed a firm pulsating mass with thrill and bruit, present in the epigastrium and measuring about 8 x 10 cm The Wasserman test *was negative An operation* was done with wiring and electrothermic coagulation of an abdominal aortic aneurysm Approximately 100 feet of wire were introduced into the aneurysm

Roentgen examination (Fig 85) showed displacement of the stomach to the left by a circular mass *with a calcified rim A* diagnosis of aneurysm of the abdominal aorta was made, based on the following features (1) the rounded, well circumscribed character of the mass (2) its location in relation to the anatomic position of the aorta, and (3) particularly, the calcification of the wall



Fig 82 B Same patient as is shown in (A) Examination 1 week later showing essentially normal appearing stomach

Marked displacement of the stomach to the left by an abdominal aneurysm as well as other classical features of its presence, is illustrated by this fully controlled case

J W, male, aged 66 During the preceding 2 years the patient had complained of dyspnea Three weeks before admission to the hospital he had had a sudden onset of dull pain in the right lower chest and in the right upper quadrant transmitted to the back He had lost weight but did not know how much Examination of the abdomen revealed a pulsating mass with systolic thrill and murmur in the epigastrium

The autopsy report was as follows "In the descending portion of the aorta at the level of the attachment to the diaphragm there is a saccular swelling approximately 10 cm in diameter The aorta passes anteriorly to this and at the level of the first lumbar vertebra there is a 5 cm longitudinal defect in its posterior wall, from which the sac originates Posteriorly, the sac is intimately



FIG 84 B Same patient as is shown in (A) Appearance of the resected omental cyst

administration of barium a soft tissue mass may be noted close to the spine Such observations however should always be supplemented by examination in the same position after the stomach has been filled with barium

disorientation At another institution it had been found that he was a luetic, with apparently luetic heart disease and thoracic aneurysm The clinical diagnosis was syphilitic aortitis with thoracic aneurysm

On abdominal examination, while no masses were palpable, there was a marked pulsation in the epigastric region synchronous with the heartbeat At autopsy, in addition to *atheromatous changes, there was a huge* aneurysm where the thoracic aorta pierced the diaphragm This aneurysm involved 18

Roentgen examination of the stomach (Fig 87) showed an irregularly outlined translucent area occupying the pars media Below this was a wide comparatively smooth defect of the greater curvature (Careful fluoroscopy was not possible *because of the patient's* serious condition) Based on the roentgen evidence, a diagnosis of malignancy of the stomach was made As noted at autopsy the *deformity was due to the localized area of* pressure of the abdominal aneurysm upon the posterior wall of the stomach

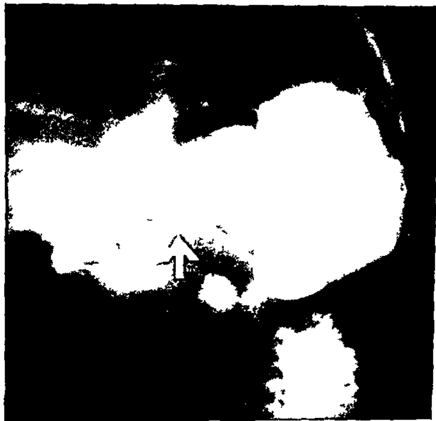


Fig 84 A Upward displacement of the stomach by a cyst of the omentum

cm of the aorta and was 15 cm in width It filled most of the pleural sac In the proximal portion of the abdominal aorta there was another aneurysm 5 cm in width and pouching out for a distance of 4 cm In the distal *portion* of the abdominal aorta there was another aneurysm, 7 cm in length and 5 cm in its widest portion

The report of the examination of the *stomach was as follows* 'The stomach is small and its rugae are prominent The mucosa is completely smooth and there are no intrinsic lesions'

When anterior displacement of the stomach by a retrogastric mass is suspected, the roentgen examination is best conducted with the patient in the left lateral position supine or erect, without and with the administration of barium by mouth With the patient on his back and a 14 × 17 cassette tangential to his left side, the ray is directed from right to left *An essentially similar* procedure is employed when the patient is erect At times in the film taken without

in association with (3) evidence of an actual pressure deformity on the posterior wall

The exact nature of the retrogastric tumor cannot be determined by this procedure While it may be due to a tumor of the body of the pancreas similar findings



Fig 86 A Displacement of the stomach by an aneurysm of the abdominal aorta

may be obtained when the lesion is a retroperitoneal sarcoma or a tumor of renal or adrenal origin

The role of the pancreas in causing displacement of the stomach will be discussed in a later chapter

The pressure of a retrogastric mass on the stomach is illustrated by the following case

J W male aged 65 During the preceding 5 weeks the patient had complained of dyspnea on exertion weakness a hacking cough and anorexia The patient coughed up ½ a cupful of red blood per day during the 10 days before admission to the hospital He had lost 35 pounds

He was a cachectic white male acutely and chronically ill markedly dyspneic and convulsed with paroxysms of coughing Examination revealed a mass occupying the upper abdomen This mass was outlined by percussion only because marked voluntary rigidity prevented palpation There was no tenderness

The autopsy report was Kidneys the right kidney weighs 130 Gm There are a few small cysts bulging on its external surface These contain clear fluid The surface is smooth and no unusual changes are noted

Engel and Lysholm [6] in their study after the administration of an effervescent mixture believed that any increase in the distance between the posterior wall of the stomach and the spine of more than the width of a vertebra was suggestive of an intervening retrogastric mass It is important to realize however, that anatomically the stomach is directed obliquely from the posteriorly placed cardia to the more anteriorly placed pylorus There is thus, a gradual increase in the distance between the spine and the stomach from the cardia to the pylorus This increase in the distance may be further enhanced by the habitus of the patient and may be much more marked in the hypersthenic and sthenic type of individual

All these factors must, therefore, be kept in mind in determining whether there is any actual forward dislocation of the stomach by a retroperitoneal mass



FIG 85 Displacement of the stomach by an aneurysm of the abdominal aorta Note calcification in the rim of the aneurysm

Of greater significance is the evidence of an actual pressure defect on the posterior wall The diagnosis of a retrogastric tumor will then depend (1) on the actual demonstration of a soft tissue mass in the left lateral films of the abdominal cavity in the region of the spine (2) displacement of the barium or gas filled stomach particularly

in association with (3) evidence of an actual pressure deformity on the posterior wall

The exact nature of the retrogastric tumor cannot be determined by this procedure While it may be due to a tumor of the body of the pancreas similar findings may be obtained when the lesion is a retroperitoneal sarcoma or a tumor of renal or adrenal origin

The role of the pancreas in causing displacement of the stomach will be discussed in a later chapter

The pressure of a retrogastric mass on the stomach is illustrated by the following case

J W male aged 65 During the preceding 5 weeks the patient had complained of dyspnea on exertion weakness a hacking cough and anorexia The patient coughed up ½ a cupful of red blood per day during the 10 days before admission to the hospital He had lost 35 pounds

He was a cachectic white male, acutely and chronically ill, markedly dyspneic and convulsed with paroxysms of coughing Examination revealed a mass occupying the upper abdomen This mass was outlined by percussion only because marked voluntary rigidity prevented palpation There was no tenderness

The autopsy report was Kidneys the right kidney weighs 130 Gm There are a few small cysts bulging on its external surface These contain clear fluid The surface is smooth and no unusual changes are noted



Fig 86 A Displacement of the stomach by an aneurysm of the abdominal aorta

The left kidney weighs 500 Gm The surface is roughly nodular and the nodules vary in size and shape but are of one general pattern —broad base with conical sometimes umbilicated surface The color of these nodules varies from whitish gray to bright yellow brown The intervening surface is gray brown in color and smooth The capsule strips with difficulty and is adherent over some of the nodules On section of the kidney the normal architecture and markings are obliterated

and there are similar smaller masses of nodes extending upwards along the aorta The mass at the hilus and that at the kidney are seen to push the stomach anteriorly There is a solid fused mass of nodes at the hilus which completely surrounds and partially constricts the inferior vena cava There is a small number of similar glands in the peripancreatic tissue

Gastro intestinal tract The esophagus stomach small and large intestine show no



Fig 86 B Same patient as is shown in (A) Note the erosion of the vertebrae produced by the aneurysm as well as the calcification of the wall

The parenchyma has been generally replaced by a mottled grayish yellow tissue found in nodules of varying sizes and also diffusely throughout the kidney from pelvis to capsule Some of these areas are soft, red brown and hemorrhagic The calyces are dilated and the mucosa is thick congested and bright yellow green in color The calyces and pelvis contain *soft yellow cheesy* material The pelvic ureteral junction appears to be blocked by a gray white fungating mass extending into the pelvis from the kidney parenchyma There is a large mass of nodes at the hilus containing material similar to that found in the kidney

intrinsic changes except for mild congestion of the stomach

Pathologic diagnosis Adenocarcinoma of the left kidney with metastases to regional and distant lymph nodes left adrenal and both lungs

Roentgenographic examination (Fig 88A) of the stomach in the usual postero anterior position showed no evidence of any abnormality Examination in the erect left lateral position (Fig 88B) showed forward displace ment of the cardiac portion of the stomach, with evidence of a pressure defect Note that the distance between the anterior wall of the

vertebral column and the posterior wall of the stomach is considerably greater than the distance occupied by the body of the vertebra It was not only the increased distance between the vertebral column and the posterior wall of the stomach but also the evidence of a pressure defect that justified the diagnosis of a retrogastric mass This forward displacement of the stomach as well as the pressure defect were also shown in Figure 88C with the exposure made with the patient in the supine position the cassette being placed against the left side of the abdomen and the ray directed horizontally through the abdominal wall from right to left

This case illustrates the anterior displacement of the stomach produced by a retrogastric mass and the technical procedure employed in the demonstration of such a lesion In this particular case the retrogastric tumor was a carcinoma of the left kidney

Forward displacement by a tumor of adrenal origin is illustrated by the next case

J R male aged 37 The patient gave a 3 weeks history of pain in the left lower chest posteriorly as well as in the left upper quadrant of the abdomen He had fever (101° F to 102 F) marked leucocytosis and a weight loss of 30 pounds



Fig 87 Deformity of the stomach by pressure from an aneurysm of the aorta

Roentgen examination of the lungs revealed tuberculosis of the right apex Examination of the sputum was negative for tubercle bacilli

Physical examination of the abdomen was essentially negative except for tenderness in the left upper quadrant

The operative findings were as follows On opening the peritoneum there was a moderate amount of clear fluid The liver and spleen were normal In the left upper quadrant retroperitoneally in the region of the left kidney there was a large hard nodular mass 15 x 10 x 10 cm In addition there were two other

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which as already noted was confirmed on surgical exploration

THE GASTRIC AIR BUBBLE

Of considerable importance is the gastric air bubble A moderate amount of air is normally present in the stomach and rising to the cardia in the erect position produces the characteristic gastric air bubble Its size and shape may show a great deal of variation because of differences in body habitus Secondly the amount of air at any given time will produce considerable change This is particularly true when air is swallowed to a pathologic degree On observing the barium being swallowed and its passage through the esophagus small amounts of air may be noted incorporated within the barium mass As the swallowing act continues the amount of air reaching the stomach may distend it to a consider able degree Rising to the uppermost por tion of the stomach the gastric air bubble becomes abnormally prominent With the act of belching and the sudden escape of air the air bubble may momentarily disappear completely or may show considerable dimi nution in size Soon with each successive swallow there is a reaccumulation of air in the stomach with a return to the original size of the air bubble That the belching of gas depends entirely on the eructation of air previously swallowed was shown by Leven[27] who analyzed the eructated gas and showed that it consisted entirely of air and that gastric fermentation played no role in the process



Fig 89 (A *Left*) Tumor of the left adrenal gland showing displacement of the stom ach to the left (B *Right*) Same patient as is shown in Figure 89 (A) Erect left lateral position showing forward displacement of the stomach by the retrogastric tumor

The gastric air bubble under certain con ditions may become chronically enlarged The left diaphragm may then be displaced upward with encroachment upon the heart and lungs Moreover such large amounts of air in the stomach may be a contributing factor in the production of gaseous disten tion of the intestine since gastric peristaltic activity may force some of the air onward

smaller masses in this region about 6 x 8 cm in diameter On opening a nodule situated anterior to the retroperitoneal tumor, the mass appeared to be of a golden yellow color resembling, somewhat adrenal cortical tissue vertently removed Pathologic examination showed this to be normal pancreas

Roentgenographic examination (Fig 89A) showed displacement of the stomach to the left by an extragastric mass

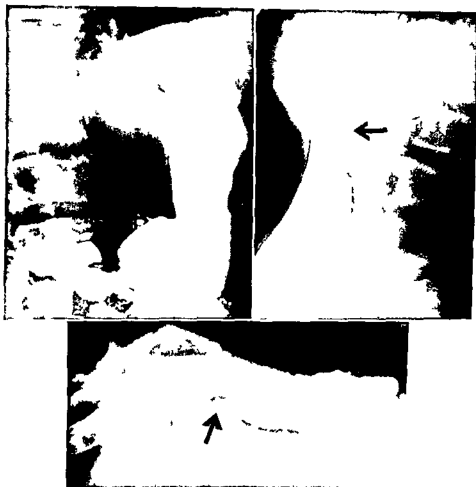


FIG 88 (A *Left*) Adenocarcinoma of the left kidney No displacement of the stomach in the posteroanterior position (B *Right*) Same patient as is shown in (A) Erect left lateral position, showing forward displacement of the proximal portion of the stomach and a pressure defect on the posterior wall (C *Below*) Same patient as is shown in (A) and (B) Examination in the left lateral supine position showing anterior displacement of the proximal portion of the stomach and the pressure defect of the posterior wall produced by the tumor mass

There was no evidence of peritoneal metastases The stomach was normal

A biopsy was taken The pathologic report was as follows malignant tumor, of undetermined type probably pheochromocytoma A piece of pancreas was also inad

Examination in the erect left lateral position (Fig 89B) showed forward displacement of the stomach and pressure on the posterior wall thus indicating that the mass was retrogastric On the basis of this evidence, a diagnosis of retrogastric tumor was made

which as already noted, was confirmed on surgical exploration

THE GASTRIC AIR BUBBLE

Of considerable importance is the gastric air bubble A moderate amount of air is normally present in the stomach and rising to the cardia in the erect position produces the characteristic gastric air bubble Its size and shape may show a great deal of variation because of differences in body habitus Secondly the amount of air at any given time will produce considerable change This is particularly true when air is swallowed to a pathologic degree On observing the barium being swallowed and its passage through the esophagus small amounts of air may be noted incorporated within the barium mass As the swallowing act continues, the amount of air reaching the stomach may distend it to a consider able degree Rising to the uppermost por tion of the stomach the gastric air bubble becomes abnormally prominent With the act of belching and the sudden escape of air the air bubble may momentarily disappear completely or may show considerable dimi nution in size Soon, with each successive swallow there is a reaccumulation of air in the stomach with a return to the original size of the air bubble That the belching of gas depends entirely on the eructation of air previously swallowed was shown by Leven[27] who analyzed the eructated gas and showed that it consisted entirely of air and that gastric fermentation played no role in the process



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through the duodenum. Occasionally, the normal duodenal bulb will show *in the* roentgenogram, an air cap surmounting it presumably derived from the air in the stomach on its way into the intestine.

Although the air bubble shows considerable variation in size and shape, it is *smooth and rounded in contour. Its shape may be altered by the presence of considerable* fluid in the stomach either as a result of hypersecretion or of the ingestion



FIG 90 Huge air bubble of the stomach. Note also the marked gaseous distention of the colon.

of food or of the opaque suspension used *in examination*. In this manner a horizontal line will mark the lower limit of the air bubble, the rest of which, however remains dome shaped. Near its upper region one may occasionally note a portion of the cardiac shadow.

The air bubble may be displaced by extragastric masses or by an overdistended colon. It may be overlapped by the splenic flexure. A more serious abnormality of the air bubble with irregularity of contour results from the intrusion of a new growth arising either within the stomach itself or, rarely *extending* into it by perforation of an extragastric mass.

An example of a huge air bubble of the stomach associated with considerable gaseous distention of the colon is illustrated by Figure 90.

GASTRIC MOTOR FUNCTION

The first attempts to determine the normal emptying time of the stomach by roentgen observation were those of Jollasse [8] in 1907, and particularly by Haudek [9] in 1912. *Although there* is considerable variation in the rate of emptying of the normal stomach the 6 hour period as established by Haudek, is clinically satisfactory.

Ordinarily, the normal stomach will have emptied completely during that time. However, the retention of small amounts of barium in the stomach beyond this time does not necessarily mean that an organic obstructive lesion is present, preventing the exit of the gastric contents. Even a residue of one fourth the barium suspension may result *from factors of a purely functional* nature. When one half or more of the barium is retained, the probability is very strong that an obstruction of the pylorus is present either as a result of organic stenosis or of spasm in the presence of an *ulcer. Twenty four hour gastric retention may be considered as definite indication* of the presence of an organic lesion. Normal emptying of the stomach within 6 hours may occur as an expression of all of the factors involved in the determination of gastric motility. It does not preclude the possibility however, of an abnormality of one of the factors which may be balanced by a hyperactivity of another. Thus spasm of the pylorus may be compensated for by gastric hyperperistalsis.

Abnormally rapid emptying of the stomach may occur as a result of exaggerated peristaltic activity or in the presence of a gaping pylorus, particularly due to rigid infiltration.

Of importance roentgenologically is the



Fig 91 Functional spasm of the stomach Note the narrowing of the pyloric portion The mucosal folds however are intact



Fig 92 Normal mucosal folds of the stomach

effect of the emotions upon the emptying of the stomach It is not at all uncommon to examine a patient for the first time and find apparent evidence of functional derangement of tone peristalsis and motor function The anxiety of the patient and the strangeness of his surroundings may contribute to this change Re examination at a later time when the patient s fears have been calmed and he is less apprehensive may show the motor manifestations to be more normal

GASTROSPASM

One of the earliest studies of gastrospasm was that of Holzknecht and Luger[20] Gastrospasm may be associated with intrinsic organic disease, or it may be a secondary manifestation from an extragastric cause, such as cholelithiasis or duodenal ulcer Autonomic imbalance may be an important factor

Gastrospasm may be of four types (1) Spasm may be limited to the pyloric ring (2) A highly circumscribed type of spasm is the incisura of the greater curvature



FIG 93 Mucosal folds in the pars cardia Note the translucent area produced by the entrance of the esophagus

Though occasionally of functional origin it may be seen opposite an ulcer of the lesser curvature and also in association with malignant ulceration In the latter condition the incisura is apt to be broader than in benign lesions and may in part be due to actual organic involvement in the malignant process (3) Large areas of spastic involvement of the stomach may be noted particularly in the pars pylorica This region may show marked constriction under fluoroscopic observation with at times complete obliteration of the entire region When filled with barium this region is smooth in contour without evidence of destruction of the normal mucosal markings This helps in differentiating the condition from malignant infiltration Moreover the contour may show variation of outline at different times The administration of atropine to physiologic effect may possibly aid in differential diagnosis by diminishing the spastic manifestations Marked pylorospasm may not necessarily be altogether functional and in some cases it may be associated with the presence of inflammatory disease of the pylorus or may be due to the actual presence of an ulcer (4) In total gastrospasm, the entire stomach partakes in the process and the appearance may simulate that of a scirrhous carcinoma of the stomach The differential diagnosis lies primarily in the inconstant manifestations in spasm as opposed to the permanent character of the deformity in malignancy, as well as in the demonstration of the intact mucosal relief when the abnormality is spastic in nature Cases of total gastrospasm have been described as a result of lead colic, nicotine poisoning and arteriosclerosis of the abdominal vessels

Illustrative Case The alterations in the contour of the stomach produced by functional gastrospasm are demonstrated by the following case

M L, male, aged 38 This patient complained of vague gastric discomfort after meals He vomited occasionally He said that he had lost 40 pounds in the last 5 years He had undergone appendectomy 27 years previously Physical examination of the abdomen was essentially negative

Exploratory laparotomy revealed no evidence of any organic lesion of the stomach or duodenum in spite of very careful examination of this region

The roentgen examination (Fig 91) revealed a persistent narrowing of the pyloric portion of the stomach The mucosal folds were intact In the absence of any organic pathology that could be discovered at operation this narrowing must be considered as functional The most important evidence as to the functional nature of the pyloric narrowing is the persistence of rugal folds and the absence of any definite irregularity of contour

THE GASTRIC MUCOSA

The appearance of the rugal folds of the stomach plays an interesting role in clinical roentgenology of this organ The folds

were generally considered to be due to a passive process resulting from the contractions of the muscular coat Forssell[31] showed that these folds had an independent mechanism that they varied in appearance from time to time because of active movements of the membrane itself throughout the entire alimentary canal, and that these alterations were conditioned by contractions of the muscularis mucosae

Thus according to the work of Forssell, factor in the changeability of the mucosal relief is the hydrodynamic action of the fluid content of the submucosa which, by its variability causes corresponding alterations in the thickness of the gastric folds The regulatory mechanism probably lies in the plexus of Meissner and in the ganglia of the muscularis mucosae

Gunn and Underhill (1915), and King and Arnold (1922) published observations on active movements of the digestive



Fig 94 Normal mucosal folds of the pars media of the stomach

the mucous membrane of the stomach is not a constant anatomic structure but is susceptible of considerable change of an intrinsic nature Nevertheless in different divisions of the dige tive tract and in various animals and perhaps in different individuals there are certain recurring local formations The lesser curvature of the stomach is more static (Waldeyer s Magenstrasse) whereas the rest of the gastric relief changes considerably The mucous membrane takes care of the fine regulation of chemical activity while the musculature is the coarse motor mechanism of the stomach Forssell believed that an important mucous membrane of the cat and dog and Thorell (1924) showed that the surviving muscularis mucosae of man and of warm and cold blooded animals has the capacity of making rhythmical movements of its own and in combination with nervous elements it responds in a completely typical way to different stimuli, such as physostigmine barium chloride papaverine and nicotine[3]

Roentgen Aspects A study of the roentgen appearance of the gastric mucosa is not new, indeed it is as old as the history of gastro intestinal roentgenology itself In the early work of Rieder[33] one may note

roentgenologic evidence of the folds of the stomach It was Holzknecht[34] however who first emphasized the importance of a routine investigation of the gastric mucosa and recommended palpatory manipulation of the stomach immediately after the ingestion of a small amount of a thin watery suspension of bismuth, in order to accomplish this purpose Following this preliminary examination, the stomach was then completely filled and a contour study was made

Special methods for the more detailed study of the mucosal relief were devised by von Elischer[35] Eisler and Lenk,[6] Chaoul[37] and Berg[8]

Rendich[9] working in the laboratory of the Roentgen Department of Bellevue Hospital, made one of the pioneer contributions to the study of gastric rugae He emphasized the value of such studies for the more accurate and detailed visualization of ulcer and the radiating folds which may accompany it for the study of the changes produced by malignancy for better visualization of the enterostomized stomach and in the study of gastritis In one normal case two roentgen exposures at an interval of 2 minutes showed an identical configuration of the mucosal pattern

Technic The following procedure is recommended for the study of the gastric mucosa For a detailed study of the mucosa to be made by the roentgen method which will approach as nearly as possible actual *anatomic conditions* it is essential that examination be conducted on an empty stomach Unfortunately, such an ideal condition is rarely ever possible and a small amount of mucus is present under normal conditions which cannot always be removed even by gastric lavage

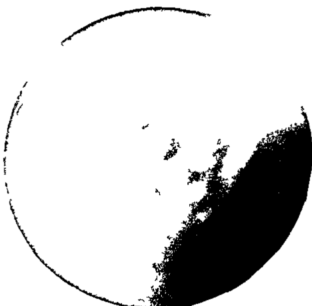


Fig 95 Normal mucosal folds of the pars media

A suspension of approximately equal parts of barium sulfate and water is employed to which a small amount of acacia may be added in order to make the opaque substance stick more intimately to the mucous membrane

Examination may be conducted with the patient in the erect or in the supine position The advantage of the supine position is that the suspension is less apt to accumulate at the lower pole of the stomach

The patient is instructed to drink the mixture one mouthful at a time The material is spread manually over the mucosal

surface The additional amount of barium which may be necessary for the patient to swallow will depend upon the individual case Frequently a surprisingly small amount of the mixture is sufficient It is rarely ever necessary to administer more than a total of 150 cc

Following the distribution of the barium over the mucosal surface in the supine position the tilt table may gradually be turned downward until that degree of inclination is obtained which permits the most uniform distribution of the suspension Varying degrees of compression may be essential in order to bring the mucosal appearance into clearer focus and by means of a spot film device prompt roentgen exposure is made

Another way in which the gastric mucosa may be demonstrated is for the stomach to be filled with a larger amount of barium sufficient for contour study The patient is placed in the prone position and turned slightly to his right in order to avoid excessive pressure against the stomach Films are taken at varying intervals as the stomach gradually empties itself of the barium suspension These films are developed promptly and further observations are made as required by the individual case An examination conducted in this manner will yield valuable information regarding the roentgen appearance of the gastric mucosa and of the external configuration of the stomach itself

A word of caution is necessary In his desire to study the mucosal relief in all its detail one must not lose sight of the fact that this is only an additional procedure which under no circumstance is to supplant the more fundamental method of studying the stomach by completely filling it The most valuable information in the examination of the stomach is still to be obtained by this procedure and mucosal studies must be considered only as an accessory aid



Fig 96 Mucosal folds of the pars pylorica

There are a number of factors which influence the roentgen appearance of the mucosal relief

Mucus present in the stomach may influence the gastric relief in two ways (1) by

producing a negative shadow within the barium relief, and (2) by becoming impregnated with barium similar to the process which has been shown to occur in the roentgen examination of the colon in the presence of mucus An exaggeration in the production of mucus may increase this tendency Moreover, if the excess mucus forms a coating on the gastric mucous membrane, there may be an absence or an apparent irregularity of the folds not due to any intrinsic derangement of the anatomic characteristics of the mucosa itself

Occasional, small globules of air may be mixed with the gastric secretion and may produce translucent areas in the gastric relief

The differentiation of translucent areas produced by mucus and air from actual intrinsic changes in the mucosa may be made in two ways (1) by the fact that such translucent areas may be moved about by palpatory manipulation behind the fluoroscope, and (2) because re examination particularly after preliminary gastric lavage *may fail to show the recurrence of* this phenomenon Similarly the presence of food in the stomach either because of improper preparation of the patient or because of retention due to pyloric stenosis may also produce translucent areas of varying sizes in the roentgenogram These may *similarly be differentiated from intrinsic* gastric pathology by inconstancy of localization under manipulation and as the result of re examination

In addition to those factors which have already been considered, such as retained gastric secretion, food, mucus and globules of air, the appearance of the mucosal relief picture may also be influenced by the thinness of the contrast mixture employed and the uniformity of its distribution Obviously the thicker the mixture employed, the greater the opportunity of covering the rugal folds in addition to the grooves between them, so that fewer folds of an apparently distorted character may appear in the roentgenogram Lack of homogeneity in the distribution of the barium may similarly be responsible for apparently bizarre relief pictures Thus if barium fails to enter a groove, the translucent areas in the roentgenogram may represent not only the actual fold itself but the width of the barium free groove in addition The quantity of barium suspension employed may *also influence the apparent size of* the folds since dilatation of the stomach may produce various degrees of obliteration



Fig 97 Normal mucosal folds of the pars pylorica

Another important factor is the degree of mechanical pressure which is exerted upon the stomach Soft, readily pliable folds may be completely obliterated by ex

cessive pressure Areas free of folds may thereby be caused in the neighborhood of a peptic ulcer which may be falsely interpreted as being due to the displacement of the barium by abnormal swelling of the folds Insufficient pressure may either fail to show the folds in a clear manner or may give the impression of haziness of outline It is also possible that improperly applied pressure may cause an artificial appearance of puckering in the roentgenogram

Still another factor capable of producing a bizarre roentgenogram is that which comes from the overlapping of anteriorly and posteriorly placed folds In the normal mucosal relief folds may be noted which are longitudinal in direction hugging the lesser curvature at the Magenstrasse Occasionally a longitudinal fold of the esophagus may be noted continuous with the fold of the lesser curvature The rest of the folds of the stomach run fairly parallel as far as the region of the incisura angularis when they diverge in a somewhat spiral manner toward the distal portion of the greater curvature Occasionally, the folds lack this divergence and run through the entire length of the pylorus in longitudinal fashion In the immediate prepyloric region the folds may occasionally run in a transverse direction There may be a convergence of folds at the entrance to the pyloric ring



Fig 98 Rounded translucent areas produced by mucosal folds in cross section



Fig 99 Prominent and tortuous mucosal folds of the stomach

Along the greater curvature the folds are more tortuous and frequently show characteristic toothlike serrations The folds and the grooves between them may show considerable variation within normal limits

Mucosal relief studies of the pars cardia may show a small rounded translucent area high up near the lesser curvature produced by the entrance of the esophagus This appearance must not be confused with a tumor Differential diagnosis depends on (1) the fact that it corresponds anatomically to the position of the entrance of the esophagus (2) verification of this fact when there is any doubt by combined examination of the barium filled esophagus and stomach and (3) changes in the size of the translucent area or its complete absence at times depending on transient variations in the degree of dilatation of the cardiac end of the esophagus where it enters the stomach

Illustrative Cases The appearance of

the normal gastric mucosa is shown in the following roentgenograms

Figure 92 shows mucosal folds throughout the stomach running, for the most part, in an essentially parallel manner There is also evidence of decussation of some of the mucosal folds

A normal pattern of the mucosal folds in the pars cardia is illustrated by figure 93 Note the irregularly rounded, translucent



FIG 100 Marked enlargement and distortion of the mucosal folds of the stomach

area high up produced by the entrance of the temporarily distended esophagus

Figure 94 shows the vertical mucosal folds of the stomach in the pars media These folds run essentially parallel to one another They exhibit a moderate degree of tortuosity

Figure 95 shows the mucosal folds at the junction of the pars pylorica and the pars media, running in a manner essentially parallel to one another A moderate degree of tortuosity may be noted, particularly near the greater curvature border

Figure 96 shows the mucosal folds in the *pyloric portion of the stomach In this area,* the tendency may be noted for the mucosal folds to be directed from the lesser curvature toward the distal portion of the opposite *site greater curvature*

The folds in the pars pylorus may at times run parallel Small, rounded translucencies in the course of the folds apparently represent bits of mucus or tiny air bubbles (Fig 97)

Some of the larger, more clearly defined rounded translucent areas represent mucosal folds in cross section (Fig 98) They may be confused with small polyps or abnormal polypoid changes Permanently present when the cause is actual organic disease, the pattern will change in repeated observations of the identical region when *the condition is caused by normal mucosal* folds

At times the folds may be unusually prominent and tortuous (Fig 99) and may *be, nevertheless, the result of functional* changes in the absence of an actual gastritis The suspicion of underlying inflammatory pathology as the cause is much stronger when the mucosal pattern shows the very marked changes to be seen in figure 100 The relation of such findings to *the diagnosis of chronic gastritis* will be discussed in the next chapter

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18

Chronic Gastritis

ETIOLOGY AND PATHOLOGY

One of the earliest descriptions of an erosive gastritis was that by Morgagni[1] in Epistle 29 No 20, of his famous work *The stomach was full of erosions some of which had become gangrenous* Some of the erosions were close together and were very small In addition to the gastric erosions he also found erosions in the proximal portion of the duodenum and in the esophagus He believed that the condition was probably due to something the patient had eaten

In Figures 1 and 2 (Plate 2 Installment 30) Cruveilhier showed beautiful examples of what he described as a follicular gastritis In the duodenum and throughout the small intestine scars were present similar in nature to those in the stomach Cruveilhier stated that follicular gastritis had not received the clinical recognition the condition deserved, although such ulcerative inflammation was a common form of gastritis He described cases in which the entire mucosa was involved in superficial erosions Hemorrhagic erosions of the stomach might be present causing hematemesis He described the appearance in a number of cases and included excellent reproductions of the autopsy findings

Rokitansky[3] recognized the existence of a gastritis mucosa associated with hypertrophy of the mucous membrane which in an advanced degree might show a warty surface leading to the formation of polyps He found chronic catarrhal inflammation to be most common in the pyloric portion of the stomach

The existence of chronic gastritis was also described in the works of Brinton[4] and Dieulafoy[5]

The serious difficulty in the study of the pathology of chronic gastritis was due to *the changes which developed post mortem* A great advance in the study of the problem resulted from the contribution of surgery which yielded fresh material obtained by partial gastrectomy

The changes produced by chronic gastritis may be seen in the fresh specimen and the characteristics may be retained if the specimen while still warm, is fixed in formalin The gastric mucosa is irregular in appearance It may show polypoid proliferations and the presence of acute inflammation with fresh or healed erosions In advanced cases the deeper tissues of the wall of the stomach may be involved with rigidity and hypertrophy and occasionally with narrowing of the pyloric antrum The serosa may show reddening with the production of adhesions and inflammatory hyperplasia in neighboring lymph nodes

ROENTGEN DIAGNOSIS

The evidence obtained on roentgen study of the gastric mucosa must be interpreted with considerable care before one can safely conclude that it necessarily justifies a diagnosis of intrinsic organic disease This conservative attitude is based upon the following observations

1 Increased prominence and tortuosity of the mucosal folds may in some cases be due to a reflex stimulation of the gastric innervation[6] The vegetative nervous system apparently plays a great role influenc

ing the behavior of the mucosal relief through its effect upon the muscularis mucosae The significance of changes in the gastric relief must therefore be interpreted in the light of the functional behavior of the patient from the standpoint of autonomic balance

2 Changes in the mucosal relief with increased prominence of the gastric folds may be the expression of an allergic response to food[7] and therefore may be the result of a functional derangement and not of organic disease

3 Abnormal prominence of the mucosal folds may be due to the presence of giant rugae of congenital origin [8] In such cases a localized, circumscribed thickening of the mucosal relief may have no bearing whatever as an indication of an actual gastritis Prominent rugae of this nature may show a pure hyperplasia of the mucosa without any evidence of inflammation

4 Particularly significant is the fact that there is no correlation between the nature of the folds, as seen roentgenologically, and their actual appearance as observed gastroscopically Only in those cases in which translucent areas in the roentgenogram correspond to nodular wartlike structures in the mucosa is the roentgenographic evidence acceptable

Unusually prominent mucosal folds may be present in the absence of any evidence of organic derangement On the other hand, in some cases in which the roentgen manifestations appear to be those of a normal mucosal relief, definite evidence of organic change of the mucosa has been found Thus, in some cases of pernicious anemia with atrophic gastritis, roentgen examination may show the presence of apparently normal gastric rugae This appears to be due to the ability of the muscularis mucosae to throw even an atrophic mucosa into folds Narrowing of the mucosal folds therefore, is not to be considered an evidence of an atrophic gastritis but is rather to be attributed to a diminution of the tone and turgor of the gastric submucosa which is associated with various degrees of dehydration

5 Still another difficulty in the roentgen diagnosis of chronic gastritis lies in the fact that, even with a painstaking technic, it is not always possible to obtain an exact anatomic representation of the gastric rugae Thus mucus, which it may be impossible to remove completely, may adhere to the grooves between folds, thereby preventing the entrance of barium Under such circumstances, folds in the roentgenogram may appear deceptively wide Moreover, a slight increase in the thickness of the barium suspension, or the application of an improper degree of compression, or the overlapping of anteriorly and posteriorly placed folds, may add to the difficulties and distort the picture In addition, adherent flecks of mucus may displace the barium, with the production of translucent areas which may mimic those produced by actual excrescences of the mucosa of a pathologic nature These technical difficulties are therefore added reasons for a conservative approach to the diagnosis of chronic gastritis on the basis of the roentgen data alone

It cannot be emphasized too strongly, therefore, that the roentgen diagnosis of gastritis is subject to considerable error of interpretation To base the diagnosis of gastritis upon apparent increased thickness and tortuosity of the rugal folds as they appear in the roentgenogram is not justified since they may be functional in nature or in rare instances, congenital in origin and not necessarily the expression of organic disease Gastroscopy in particular has called our attention to the danger of reading the presence of organic disease into a gastric relief of this nature Just as prominence of the mucosal folds in the roentgen relief does not necessarily justify the diagnosis of a hypertrophic gastritis, so an apparent diminution in the size of the folds does not justify the diagnosis of an atrophic gastritis Moreover, definite pathologic evidence of gastritis is not incompatible with an apparently normal size and distribution

of the mucosal folds in the roentgen relief

Since gastritis is a comparatively frequent disease it is obvious that the roentgen diagnosis of chronic gastritis will often appear to be corroborated though this may be simply due to the law of chance In the diagnosis of chronic gastritis gastroscopic study is apparently far more reliable in general than the most painstaking and careful roentgen technic

Are there any roentgen features which may justify the assumption that an actual chronic gastritis is present? The most reliable finding is that of small, fairly well rounded translucent areas of a permanent character representing actual wartlike excrescences or polypoid changes Here too however one must be certain that such translucencies are really permanent since clumps of mucus or small particles of retained food may produce similar changes However, such alterations are of a tempoary character and palpatory manipulation under fluoroscopic control as well as reexamination should demonstrate their evanescent nature

Spriggs and Marxer[9] described 19 cases of polypoid hyperplastic swelling of the gastric mucosa with correlation of the roentgen changes and the anatomic appearance of the lesion

While unusually prominent and tortuous mucosal folds are not necessarily the result of a pathologic process extreme exaggeration of these folds associated with a lack of pliability may furnish evidence of a suggestive nature This is illustrated by the well authenticated case reported by Cole[10]

At times the infiltrative character of the hypertrophic gastritis may produce changes in the roentgen appearance of the stomach indistinguishable from malignant infiltration The occasional difficulty in differentiating between the deformity produced by chronic gastritis and carcinoma is illustrated by the carefully controlled studies of Freedman Glenn and Laipply[11] and the well documented report by Hinkel[12] In Hinkel s case both the roentgen evidence of deformity of the pars cardia and the gross appearance of the resected specimen (photographs of which are included in the report) were certainly indicative of an intramural tumor Microscopic examination showed an adenomatoid hyperplasia of the mucous glands with chronic diffuse gastritis

In a desire to correlate the roentgen findings in the mucosal relief with evidence of organic change, one must not lose sight of the fact that alterations of contour in the completely filled stomach may also be noted in the presence of chronic gastritis The irregularity of contour may be demonstrable in two ways (1) Both the lesser and greater curvatures may appear extremely jagged The fact that the lesser curvature is involved is of great significance since the greater curvature itself may often exhibit some degree of irregularity due to the normal distribution of the mucosal folds in this region (2) Various degrees of irregular narrowing not of a jagged character may distort the contour particularly in the antral region These changes may be associated with various degrees of narrowing and stenosis of such nature as actually to make hazardous or impossible a differentiation from malignant infiltration Superadded spasm may further distort the picture and exaggerate the difficulties of differential diagnosis

An extremely rare type of abnormality which may develop in association with inflammatory changes of the stomach is that of emphysema The condition may be recognizable roentgenologically during life Groups of small bubbles may be noted outlining the contour of the stomach[13]

Illustrative Cases The following is a case of hypertrophic gastritis which was verified surgically

J L male aged 29 The patient had complained of epigastric pain for the past 7 years There were periodic remissions During the 6 weeks before his admission to the hospital he had considerable pain which came on at any time and occasionally at night This pain was

relieved by milk and alkalies He had vomited on four occasions in the preceding month He had lost 50 pounds in the previous 2 years

Gastric analysis revealed a persistent absence of free hydrochloric acid

At operation, the stomach was considerably enlarged The duodenum was normal There was marked induration of the lesser curvature of the stomach extending from the pyloric portion up to the cardiac portion Enlarged soft nodes were found in the lesser and the greater curvatures Gastric resection was performed

The report on the macroscopic examination was as follows 'The specimen is a stomach received in formalin It measures 6 inches along the lesser curvature and 9 inches along the greater The serosa is smooth and glistening There are a few large, firm lymph nodes along the lesser curvature, which on section show haemorrhagic centers The stomach was opened along the greater curvature revealing a greatly hypertrophied mucosa The folds are so thickened and elevated as to give the mucosa the appearance of the outer surface of the brain At the cardiac end, along the lesser curvature, the hypertrophied mucosa forms an elevated circular mass about 2½ inches in diameter The cardiac end of this mass appears to be ulcerated down to the muscularis One small area near the pyloric end of the mass shows a superficial erosion with discoloration in the surrounding tissues



Fig 101 (A *Left*) Chronic gastritis Note the irregular contour of the lesser and greater curvatures and the translucent areas at the pars cardiaca (B *Right*) Same patient as is shown in (A) Note the small translucent areas in the pars media corresponding to prominent gastric rugae Proximal to this area, there is evidence of a polypoid mass hugging the lesser curvature and an irregularly outlined collection of barium at its apex

For the microscopic examination, three sections were taken from the mucosa, one from the greater curvature at the cardiac end one from the polypoid mass at the lesser curvature at the cardiac end and one from near the pylorus All showed a thickened mucosa with hyperplasia of the glands edema and lymphocytic infiltration The section from the greater curvature at the cardiac end showed epithelial infiltration of the muscularis The infiltrating glands were cystic well circumscribed and lined with several layers of poorly defined cuboidal cells The lumina of these cysts contained a few large circular pale cells with hyperchromic oval nuclei and a few polynu

clear cells In the section through the ulcer from above down there was a layer of necrotic tissue one of granulomatous tissue one of muscle and finally a subserosa heavily infiltrated with lymphocytes A section *through a lymph node at the cardiac end* along the lesser curvature showed lymphocytic hyperplasia There were several areas of lymphocytes and plasma cells infiltrating the connective tissue of the node There was no evidence of malignancy

ticularly marked in the proximal portion of the pars media Prior to operation the diagnosis was of an ulcerating lesion of the lesser curvature pars cardia probably malignant, associated with a hypertrophic gastritis *As noted in the examination of the* resected specimen (Fig 101C) the essential pathologic lesion was a hyperplastic gastritis with hypertrophic mucosa forming an oval mass in the pars cardia at the lesser curvature border of which there was an ulcer



Fig 101 C Same patient as is shown in Figure 101 A and B Specimen removed at operation Note the centrally located polypoid mass and the unusually prominent gastric folds

The diagnosis was gastric ulcer—hyperplastic gastritis

Roentgen *examination revealed the following* The stomach was considerably increased in size There was an ovoid translucent area at the pars cardia near the lesser curvature with an absence of normal mucosal folds Barium was present however at the lesser curvature border of this region The entire lesser curvature of the stomach (Fig 101A) exhibited a marked jagged irregularity which was persistent In Figure 101B the mucosal relief showed numerous small *translucent areas throughout* These were par

The essential roentgen findings in the diagnosis of the hypertrophic gastritis were the *toothlike irregularity of the contour of the* stomach and the mucosal relief with atypical mucosal structure and the translucent areas produced by the hypertrophic folds

The extent to which a chronic gastritis may produce a deformity in the contour of the stomach is illustrated by the following case

E S male aged 34 This patient was ad*mitted to the hospital* with the chief com

plaint of cough, fever and chill of 3 days' duration He had had some epigastric pain for the past 2 weeks A diagnosis of pneumonia involving the left lobe was made at that time About 2 weeks after admission, he vomited blood following an attack of severe epigastric pain He then stated that he had had several similar attacks 2 years previously

Physical examination revealed the abdomen to be tender and spasm was noted in the midepigastric region The Wasserman reaction was negative

Fractional gastric analysis revealed complete absence of free hydrochloric acid

vasion The glands appear normal There is no invasion beneath the muscularis mucosa No evidence of malignancy is present"

Examination of a lymph node of the mesentery revealed the following 'The general architecture is fairly well preserved, but there is hyperplasia of the reticulo endothelial cells The germinal centers share in this hyperplasia to a slight extent Some of the sinuses are dilated and contain reticulo endothelial cells There is no evidence of metastatic malignancy"

The diagnosis was reticulo endothelial hyperplasia of the lymph nodes

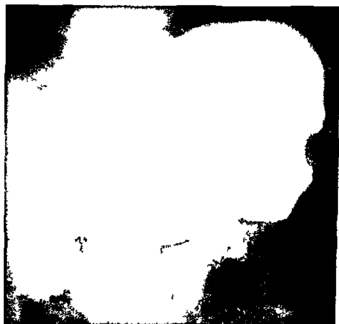


Fig 102 Chronic gastritis

The operative findings were 'The stomach is of average size There are no gross masses The wall of the stomach feels more firm and thickened than usual especially at the distal end in the prepyloric region There are several soft but definitely enlarged lymph nodes in the mesentery and greater omentum The liver is normal in size and consistency No palpable masses are present in the liver Resection of the distal third of the stomach was done

Pathologic examination of the resected stomach revealed the following The specimen consists of the distal third of the stomach The mucosa is intact and the wall feels thicker than normal However, no evidence of malignancy is noted Microscopic examination of the section reveals thickened mucosa and some fibrosis of the wall with some round cell in-

Roentgen examination (Fig 102) revealed a persistent narrowing of the distal portion of the stomach The degree of narrowing was most marked in the immediate prepyloric region There was a smooth broad defect of the greater curvature pars media with a superficial irregularity of the greater curvature distal to this area The pyloric ring was normal The duodenal bulb was elongated but was otherwise normal The preoperative diagnosis was an infiltrative lesion involving the distal portion of the stomach the most likely cause being thought to be scirrhous carcinoma As determined by both macroscopic and microscopic study the cause of this deformity was a chronic hyperplastic gastritis

The next case is an example of the marked degree of deformity of the stomach

which may be produced after swallowing lye simulating the deformity of a malignant lesion

C T male aged 20 Fifteen days before admission to the hospital the patient awoke in the morning with a nosebleed He drank a glass of what he thought was water and immediately thereafter he had a sharp burning pain in his throat and stomach following which he vomited blood He had difficulty in swallowing thereafter and 5 days later vomited an esophageal cast (proved microscopically) Visualization of the larynx and the esophagus showed ulceration throughout It was later learned that the fluid which the patient had imbibed was lye

Esophagoscopy later revealed numerous ulcerations combined with esophagitis Five days after his admission to the hospital the patient vomited practically all his solid and liquid food

At operation marked thickening was found in the pyloric region this had apparently produced a partially obstructing lesion A biopsy was taken from the stomach wall

The microscopic examination showed the mucosal surface to be covered with an exudate of clotted blood cellular debris and polymorphonuclear and mononuclear cells The mucosa itself was eroded in many places The glands showed a uniform granular degeneration in many places reaching complete disorganization although the bases of the glands were well preserved

A gastroenterostomy was done as well as a gastrostomy

Roentgenographic examination about 1 month after the operation revealed the following



Fig 103 Narrowing and obstruction of the distal portion of the stomach due to the swallowing of lye

1 An extreme narrowing of practically the entire esophagus of the type seen as a result of chemical corrosion

2 Examination of the stomach (Fig 103) showed practically complete occlusion of the pylorus with a funnel shaped appearance of the stomach proximal to the area of constriction There was a gastroenterostomy with only a small amount of barium leaving the stomach The gastrostomy tube was also noted The appearance of the stomach was not at all that usually seen as a result of an infiltrative lesion The history, the findings at

operation and the pathologic examination of the biopsy specimen all showed that the pyloric occlusion was the result of a severe gastritis due to lye

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19

Hypertrophy of the Pylorus

ETIOLOGY

ROENTGEN DIAGNOSIS

ETIOLOGY

The subject of hypertrophic stenosis of the pylorus is of considerable clinical and roentgenologic importance There are a number of causes which may underlie the development of a condition of this nature and a survey of these factors will aid considerably in the interpretation of the roentgenologic findings in such cases

1 Hypertrophic pyloric stenosis may be of congenital origin One of the earliest descriptions of pyloric occlusion coming under this classification was given by Calder[1] In addition to the fact that this condition is present at birth however it is important also to realize that although of congenital origin such stenosis may last well into adult life Thus Maier described the autopsy findings in 31 such cases the ages of the patients varying from 12 to 80 years There was a history of epigastric pain and vomiting going back to early infancy The stomach was dilated the antrum funnel shaped and narrowed and a constriction 2 to 8 cm in length represented the pyloric canal The pyloric sphincter was hypertrophic and in some cases the rest of the stomach as well Heaped up folds in the pyloric canal were an additional factor in exaggerating the obstruction There was no evidence of ulceration or inflammation

2 Another cause of hypertrophic pyloric stenosis is an underlying gastritis In such cases the narrowing is due to an actual inflammatory hyperplasia involving the mucosa and submucosa associated with hypertrophy of the muscular layer the entire pathologic process leading to various degrees of pyloric occlusion

3 The presence of an ulcer particularly at the pylorus may lead to stenosis as a result of an associated chronic gastritis exaggerated by spasm ultimately leading to thickening of the pyloric muscle This tendency for the pyloric muscle to become hypertrophied secondary to ulcer was shown by the anatomic investigation of Horwitz Alvarez and Ascanio[3] They found the pyloric muscle in 47 individuals without organic disease of the stomach or duodenum to vary from 3 8 to 8 5 mm in thickness It generally became hypertrophied in the presence of gastric ulcer In some cases the ulcer as the initiating factor may disappear and the tendency to pyloric hypertrophy may then proceed of its own momentum

4 The possibility must also be considered that spasm of extragastric origin may also be a factor in the development of pyloric hypertrophy Thus Thomas and Wheelon[4] showed the reflex origin of pylorospasm secondary to stimulation of afferent nerves of various viscera Pylorospasm of extragastric origin has long been recognized as arising on the basis of such lesions as gallbladder disease The role of the autonomic system in the production of antral spasm was previously referred to in the description of the experimental work of Klee Such spasm of long standing may well be a factor in the eventual development of pyloric hypertrophy The original cause may subside but the pyloric hypertrophy may remain

operation and the pathologic examination of the biopsy specimen all showed that the pyloric occlusion was the result of a severe gastritis due to lye

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producing irregularly outlined translucencies in the involved segment In such cases particularly of an advanced character differential diagnosis from a malignant infiltration may be difficult or impossible The obstruction may be so marked that only a thin stream of barium may be noted leaving the pylorus insufficient for visualizing the duodenum and considerable gastric dilatation may ensue

When an underlying ulcer is associated with the pyloric hypertrophy it may at times be demonstrable as a niche in the prepyloric region or on the posterior wall when the thin layer method of examination is employed Often however the ulcer itself particularly when present on the posterior wall will go unrecognized and only the organic narrowing of the pylorus be seen These changes will be similar to those in cases in which chronic gastritis is the underlying cause Indeed it is this associated gastritis rather than the ulcer itself that may be responsible for the roentgen manifestations

Essential Roentgen Findings The essential roentgen findings in pyloric hypertrophy are therefore

1 Elongation of the pyloric canal

2 Persistence of some degree of peristaltic activity, even though this may be very superficial in character

3 An intact mucosal relief

4 In some cases a concave pressure deformity of the base of the bulb when this region is demonstrable

5 Secondary gastric enlargement with disturbed motor function

6 In borderline cases however as stated above the deformity in the contour of the pylorus and the abnormal appearance of the pyloric mucosa may be of such nature as to strongly suggest the presence of malignant disease





FIG 104 (A *Left*) Congenital pyloric stenosis Appearance after filling the stomach with barium (B *Right*) Same patient as is shown in (A) Appearance 6 hours later Note the marked gastric retention

Illustrative Cases The following case is an example of hypertrophic stenosis of the pylorus

R C a 6-week old colored male child had been well and gaining weight until he was just under 3 weeks of age when he began to

ROENTGEN DIAGNOSIS

In the congenital variety in the newborn, roentgen examination will disclose marked enlargement of the stomach and particularly abnormal delay in emptying An almost complete retention of barium may be present at 6 hours, and frequently also at 24 and 48 hours and even longer The pyloric canal, when visualized, will appear markedly elongated Menwissen and Sloof[5] showed that although in normal children the pyloric canal in its longest dimension averages only from 1 to 2 mm, in infants with congenital stenosis the pyloric canal may show elongation by roentgen examination beyond the limits of 6 or 7 mm

A combination of marked gastric enlargement with abnormal retention of a marked degree in a newborn infant will therefore justify the diagnosis of hypertrophic pyloric stenosis

Differentiation from the obstruction produced by duodenal atresia should offer no difficulty In this latter condition, not only is the stomach markedly dilated but the duodenum also shows enormous distention with a failure of any of the barium passing distally The pyloric region itself shows no evidence of constriction In congenital hypertrophic pyloric stenosis the narrowing involves the distal segment of the pylorus The duodenal bulb is ordinarily not visualized because of the marked pyloric obstruction When a small amount of barium is finally forced through into the bulb, its size will appear normal Moreover while the obstruction in duodenal atresia is complete in congenital stenosis traces of barium will ordinarily be noticed to have escaped into the small intestine in the course of 24 hours

In the adult type of pyloric hypertrophy the appearance will vary with the nature of the underlying cause In the absence of chronic gastritis or ulceration the area of pyloric narrowing will be elongated and smooth in outline The mucosal folds will appear intact and some evidence of change in contour may be noted as the result of peristaltic activity Because of the partial invagination of the hypertrophied pylorus into the duodenum, there may be a concavity at the base of the bulb, as described by Kirklin and Harris[6] Such evidence of a concave appearance at the base of the bulb must, of course, be of a permanent character, since this may be observed on a single film even in normal patients The significance of such a finding is considerably reinforced by an elongated narrowing of the pars pylorica of a persistent nature

In the case of hypertrophy of the pylorus described by Crymble and Walmsley, pathologic examination showed that the cause of the tumor at the pylorus was hypertrophy of the circular muscle limited to the pyloric canal This was entirely separated from the normal pyloric sphincter, which did not partake in the hypertrophy In view of this observation, Twining[8] believed that a radiologic aid in the recognition of this condition would be an indentation to either side of the pyloric canal marking the cleft between the pyloric sphincter and the hypertrophic prepyloric muscle Roentgen evidence of this sharp delimitation between the pyloric sphincter and the localized area of pyloric hypertrophy is rather illusive and difficult to demonstrate with any degree of assurance

As a result of the obstruction resulting from pyloric hypertrophy various degrees of gastric distention and abnormal retention will result If the obstruction is marked, an insufficient amount of barium may be forced through the pylorus for proper visualization of the duodenal bulb

In the presence of chronic gastritis further difficulties are introduced into the roentgen diagnosis of the pyloric hypertrophy resulting therefrom In the first place there is a greater tendency for the contour of the narrowed pyloric region to be irregular in outline In addition, considerable change in the appearance of the gastric mucosa may result, due to the abnormal prominence of the mucosal folds and the presence of polypoid excrescences

The pathologic diagnosis was congenital hypertrophic pyloric stenosis

The roentgen examination showed that the stomach was of increased size but there was no deformity of contour The pyloric ring and duodenal bulb could not be visualized No barium was noted leaving the stomach throughout the examination (Fig 104A) At the end of 6 hours (Fig 104B) the stomach appeared even larger than at the preceding examination the pyloric portion extending over to the extreme right Only a comparatively small amount of barium escaped from operation the pyloric ring was found to be hard and cartilagenous (hypertrophic pyloric stenosis) A Ramstedt operation was performed

The roentgen examination (Fig 105) showed a hugely dilated stomach with most of the barium present distally and the rest of the stomach outlined mainly by air None of the barium had escaped into the small intestine The greater curvature of the stomach appeared to be unusually prominent probably because of the thickening of the wall Peristaltic waves were noted at the borders of both



Fig 106 C Same patient as is shown in Figure 106 (A) and (B) Pyloric stenosis Note the narrowing of the pylorus and the cup shaped base of the duodenal bulb

the stomach and was present in the small intestine

The roentgen diagnosis was congenital hypertrophic pyloric stenosis

The stomach may become comparatively huge as the result of a congenital hypertrophic pyloric stenosis

G E female aged 10 weeks This colored baby girl began vomiting at the age of 6 weeks From then on she vomited practically after every feeding Marked peristalsis was noted passing from left to right in the upper abdomen A small hard olive shaped area was palpated in the right upper quadrant At

the lesser and greater curvatures The diagnosis was congenital hypertrophic pyloric stenosis

The following case of pyloric stenosis developed apparently on the basis of a hypertrophic gastritis

C S male aged 38 The main complaint of this patient was vomiting after meals On one occasion he recognized food eaten the day before No blood was present in the vomitus The patient had lost about 30 pounds during the few weeks preceding his admission to the hospital Five years previously he had had an episode of upper abdominal distress Physical

vomit his feedings The vomiting occurred about a half hour after meals at first but at the time of hospitalization it occurred from any visible peristalsis A small mass about 1 by 2 cm was palpable in the right upper quadrant



FIG 105 Hypertrophic pyloric stenosis Note the marked gastric enlargement Most of the barium has been forced into the distal portion of the stomach

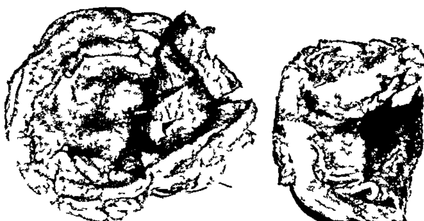


FIG 106 (A *Left*) Same patient as is shown in (B) and (C) Appearance of the resected pylorus Note abnormally prominent rugae (B, *Right*) Same patient as is shown in (A) and (C) Specimen in cross section showing abnormal thickening of the wall of the pylorus

10 to 20 minutes after feedings It was projectile in character No blood was ever seen in it and there was never any blood in the stool

On physical examination, the outline of the stomach could not be made out nor was there

At operation there was a firm tumor, 2 cm in diameter at the pylorus An incision 1 inch long was made through the circular muscle of the tumor down to the mucosa The muscle was spread with forceps and the mucosa was allowed to bulge into the opening

evidence to predetermine the exact nature of the organic lesion producing the obstruction

The outstanding roentgen features in this mal retention (2) *the elongated, narrowed,* smoothly outlined pylorus and (3) the projection of the prepyloric segment into





FIG 107 (C *Top*) Same patient as in Figure 107 A and B showing the presence of interstitial gastritis (D *Bottom*) Same patient in Figure 107 A B and C showing marked tubular narrowing at the pylorus

case upon which a diagnosis of benign pyloric stenosis might be based are (1) the evidence of gastric enlargement and abnor

the base of the bulb producing a *crescentic* appearance

The following is an example of hyper

examination of the abdomen revealed an occasional peristaltic wave in the region of the stomach No masses were palpable

At operation, a hard, nodular mass about the size of a golf ball was found at the pylorus No nodes were felt in the gastrohepatic ligament The stomach was dilated The liver was normal There was no free fluid in the abdominal cavity A partial gastrectomy was done

The report of the macroscopic examination was, "The resected specimen reveals the rugae to be prominent and edematous ' grated (in preparation), but shows definite hyperplastic changes and no evidence of malignancy Diagnosis Edematous and congested pylorus '

The prominent rugal folds and the thickened wall of the resected pylorus are shown in Figures 106A and B

On roentgenographic examination (Fig 106C) there was a marked annular narrowing of the immediate prepyloric region The stomach showed considerable secondary gastric enlargement Examination of the duodenal bulb showed no evidence of intrinsic disease (Note however, the cup shaped depression of the base of the bulb into which the narrowed pylorus appears to project) There was a gastric residue at 24 hours amounting to one half of the barium The diagnosis was pyloric obstruction of organic origin Although the pathologic evidence is that of a benign process producing the obstruction it would be impossible, on the basis of the roentgen



Fig 107 (A *Top*) Appearance of the resected specimen in pyloric stenosis Note the presence of an ulcer (B, *Bottom*) Same patient as in (A), showing marked thickening of the pylorus

The report of the microscopic examination was as follows Microscopic examination of the pyloric end of the stomach shows as the outstanding feature very marked edema of the mucosa with increase of vascularity The mucosa is also congested, except at the tips of the villi but otherwise shows no changes The epithelium shows in areas many goblet cells, distended with mucin Examination of a lymph node shows lymphoid tissue greatly disinte-

above showed that the pyloric narrowing was due to a benign hypertrophic process associated with a pyloric ulcer Roentgenologically it would be impossib e to determine the exact nature of the pathologic process since the appearance in the roentgenogram might well have been produced by a malignant infiltration

The next case of pyloric stenosis shows many features of interest

W B aged 52 This patient had been complaining of recurring attacks of pain localized to the epigastrium coming on 1 hour before mealtime and relieved by the ingestion of food There was no belching vomiting or sour eructation There had been only slight loss of weight during the 11 months of observation prior to surgical intervention He was put on the Sippy regimen and made an excellent response only to have his symptoms recur within 9 months The physical examination was essentially negative

At operation a partial gastrectomy was performed On inspecting the specimen an ulcerated area of the pylorus was found by the surgeon this he felt might be either an ulcer or an early carcinoma but more likely the former This was confirmed by pathologic examination Because of the roentgen diagnosis I am quoting the pathologic report in full



Fig 108 B Same patient a is shown in Figure 108 A Microscopic appearance



Fig 108 C Same patient as is shown in Figure 108 A and B Pyloric stenosis

trophic pyloric stenosis based on an underlying gastritis associated with ulcer

M N, female, aged 54 This patient had recurrent attacks of vomiting of 4 weeks duration There was no pain She had lost 25 pounds in the 4 weeks preceding her admission to the hospital The Wassermann was negative The physical examination was essentially negative

At operation, a firm even stony hard infiltrating mass along the lesser curvature of the stomach was found, posteriorly from the midportion of the stomach to and apparently involving the pyloric region A few small, firm nodes were present in the prepyloric region posteriorly The stomach was only moderately dilated The liver was smooth and glistening A partial gastrectomy was done

Macroscopic examination of the specimen revealed the pylorus to be practically occluded because of its greatly thickened walls Immediately next to the pylorus in the greater curvature was a well defined yellowish white dense depressed smooth diamond shaped area resembling an ulcer 1 5 by 1 cm in size (Fig 107 A and B) Adjacent to this area the mucosa was reddish brown A section through the ulcer showed the overlying tissue to be dense, white and homogeneous

The report of the microscopic examination (Fig 107C) was as follows Section through the ulcer shows the base of the ulcer to be covered with necrotic fibrin and cellular elements, beneath which is seen a rather marked vascular, fibroblastic reaction, which extends down to the muscularis This area is also infiltrated with many polys, plasma cells and eosinophiles The edge of the ulcer shows atrophic mucosa in which a similar cellular reaction is present Section through the pylorus of the stomach shows atrophic glandular elements, with marked interstitial fibroblastic proliferation vascularization and cellular infiltration similar to that described above The subjacent tissue consists of moderately vascular dense fibrous tissue The muscularis in this region shows a marked round cell infiltration between the bundles Section taken through the serosa and including the muscularis along the lesser curvature shows a similar marked cellular reaction to be present "



Fig 108 A Appearance of the resected specimen showing an ulcer in a case of marked pyloric narrowing

The diagnosis was chronic interstitial gastritis with superficial simple ulceration of the stomach there was no histologic evidence of malignancy

The roentgen examination revealed a marked annular narrowing of the pyloric portion of the stomach of fairly smooth contou There was marked obstruction to the flow of barium beyond this region The stomach was of increased size The conclusion was 'pyloric obstruction of organic origin due to an infiltrating tumor (Fig 107D)

The pathologic examination, mentioned

be present even though the ulcer is some distance away as noted in the next case

G S male aged 53 The patient gave a 1 year history of intermittent attacks of epigastric pain coming on 1½ hour after meals lasting from 4 to 5 hours and associated with occasional vomiting There was no blood in the vomitus or stool There was only slight weight loss The pain persisted in spite of medical treatment Physical examination of the abdomen was essentially negative

The operative findings revealed an ulcer on the lesser curvature about 5 cm proximal to the pylorus The gastrohepatic omentum in this area was thickened and edematous There was a marked thickening around the pylorus but no definite ulcer This thickening had caused considerable pyloric stenosis

A partial gastrectomy was done and the distal two thirds of the stomach removed

The pathologic report was gastric ulcer no malignancy pyloric hypertrophy

Roentgen examination (Fig 109) revealed a niche in the lesser curvature at the junction of the pars pylorica and the pars media with the roentgen characteristics of a benign ulcer In addition there was a tubular narrowing of the prepyloric segment of essentially smooth contour without any very sharp demarcation from the rest of the stomach interpreted as due to associated pyloric hypertrophy

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The report of the macroscopic examination was "Specimen is a portion of stomach It measures 65 mm in length and varies in width from 35 to 60 mm On opening this portion of the stomach, the interior appears negative except for one oval area measuring 22 mm in length and about 8 mm in width This is an ulcerated area about 5 mm in depth and showing a smooth base The remaining mucosa appears negative The wall of the stomach corresponding to this ulcerated area is slightly thickened and shows a small amount of fibrosis (Fig 108A) "

The microscopic report was as follows Sections of the tissue reveal a portion of stomach wall showing an area of ulceration where the mucosa submucous layer and a portion of the muscular coat are absent The surface of the ulcerated area is covered by a large amount of necrotic granular material The tissue around the ulcerated portion shows extensive fibroblastic proliferation with numerous foci of lymph follicles lymphocytes plasma cells and eosinophiles Diagnosis peptic ulcer (Note there is no evidence of malignancy in this tissue) (Fig 108B)

Roentgenologically a diagnosis of carcinoma of the pylorus had been made, based on the marked irregular narrowing of the antrum persistent throughout prolonged fluoroscopic examination and on all films In addition several translucent areas were noted in this region which suggested destruction of the mucosa due to new growth The roentgen appearance was evidently the result of the extensive fibroblastic infiltration that was reported by the pathologist (Fig 108C)



Fig 109 Hypertrophy of the pylorus associated with gastric ulcer

Hypertrophic stenosis of the pylorus may

supply the distribution of the lymphoid follicles may also play a part in the greater susceptibility of certain regions of the stomach and duodenum to the formation of ulcers Such follicles are much more common in the pyloric portion of the stomach than in the cardiac region They also are more frequently encountered on the lesser curvature than on the greater curvature and also predominate in the duodenal bulb An abscess developing in such a follicle may perhaps be a factor in the eventual origin of an ulcer as originally suggested by Cruveilhier

Aschoff[4] evoked a mechanical factor in his endeavor to explain the localization of ulcer in the region of the lesser curvature He described two types of mucosal erosions those in the fundus and those along the gastric pathway or ' Engpasse of Forssell Whereas the fundal erosions healed rapidly chronic ulcers practically speaking developed only from the second type of erosions In experiments on rabbits by Dr Yano which Aschoff recorded losses of substance produced by burns on the gastric pathway healed with much more difficulty or not at all while in the fundus the artificially produced erosions healed fairly rapidly As a result although only a faint scar remained to mark the site of a fundal erosion an actual chronic ulcer might develop in the gastric pathway Aschoff believed that an erosion of the Engpasse is in more direct contact with the acid gastric juice and is subjected to greater mechanical injury by the peristaltic motion in that region and also that the protective mucus which is secreted in the fundus and is an aid in healing is generally not present in the gastric pathway

On the basis of such conceptions Aschoff found it necessary to invoke a mechanical factor in the structural and functional peculiarities of the Engpasse to explain the development of chronic ulcer from a mucosal erosion A survey of these various factors gives us a clearer insight into the reasons why roentgenologically peptic ulcer is so commonly found at or near the lesser curvature of the stomach

Sturtevant and Shapiro made a careful analysis of 7 700 autopsy records at Bellevue Hospital during the period from 1904 to 1922 They found 120 gastric ulcers of which 34 were healed In this same series were 44 duodenal ulcers of which 9 were healed Of the 86 cases of open gastric ulcer there were 4 in which an open duodenal ulcer was also present Since these were also included in the series of open duodenal ulcers there was thus a total of 117 cases of unhealed gastric or duodenal ulcer or both Two per cent of the total of autopsy cases in the Bellevue Hospital series showed the presence of a gastric or a duodenal ulcer, either active or healed Gastric ulcers represented 1 5 per cent of the total

Fenwick, on the basis of 47 912 autopsies from various sources mainly continental found 4 2 per cent of gastric ulcers William H Welch arrived at an estimate of 5 per cent based mainly on continental reports although in 800 autopsies at Bellevue Hospital he found only 6 gastric ulcers Lockwood also found 6 gastric ulcers in 1 000 autopsies at Bellevue Hospital and Harlow Brooks reported 9 cases in another series of 1 000 at the same institution

Sturtevant and Shapiro found combined gastric and duodenal ulcer five times in a series of 159 cases of ulcer This represented an incidence of about 3 per cent Of the duodenal ulcers 11 per cent had a coexisting gastric ulcer Of the 120 gastric ulcers 86 were single the rest were multiple there being 12 in one patient They quoted Berthold who described a patient who had 34 ulcers and the patient cited by Lange in whom the ulcers were so numerous they could not be counted Of the 120 gastric ulcers 21 were active Five acute ulcers were found in infants In 49 cases of simple gastric ulcer the size of the ulcer varied from less than 0 5 cm to 5 cm by 6 cm The authors found that as ulcers increase in number they are apt to decrease in size

Of the total number of gastric ulcers 9

20

Gastric Ulcer

Although descriptions of gastric ulcer checked at autopsy can be found in the literature from the time of Marcellus Donatus in 1586, it was not until the classic descriptions of Morgagni[1] Abercrombie and particularly Cruveilhier[3] in 1829, that simple chronic ulcer of the stomach became clearly recognized as a definite pathologic entity

Cruveilhier s first patient was a coal carrier 23 years old who was brought to the hospital in a moribund state on December 15 1829 The preceding morning while carrying a bag of coal he had experienced a sudden attack of excruciating abdominal pain He died 3 hours after his admission to the hospital The autopsy revealed the cause of his death to have been the perforation of a round ulcer of the stomach at the pyloric orifice Cruveilhier also included other examples of characteristic simple chronic ulcers of the stomach One of his pictures was of an unusually large gastric ulcer which had perforated through all the coats of the stomach, exhibiting a considerable portion of the pancreas at the floor of the ulcer

Cruveilhier s description of chronic simple ulcer of the stomach was epoch making Such an ulcer according to him consists of a loss of substance usually circular and of punched out appearance with a grayish base It is of varying size is almost always solitary, and is usually situated either at the lesser curvature or on the posterior wall of the stomach It burrows through the walls of the stomach and if this process is not walled off by the formation of adhesions, the ulcer goes on to perforation with the escape of gastric contents into the peritoneal cavity In his opinion the primary lesion was an erosion of the mucosa due to the ulcerating inflammation described by Hunter Finally, this erosion becomes a true *ulcer with all its pathologic characteristics* Cruveilhier also stated that simple ulcer of the stomach shows none of the attributes of cancer—a fact that is best demonstrated by its curability and tendency to heal under simple therapy Essentially his description of the pathology of gastric ulcer holds to this very day

LOCALIZATION AND FREQUENCY

A factor which may explain the predilection of ulcer for the lesser curvature of the stomach may be the nature of the circulation in this region The blood vessels are smaller here and make fewer anastomoses Similarly in the bulb it is stated the arteries are few in number and small in size and do not anastomose as freely as elsewhere in the duodenum Such peculiarities may possibly make them more susceptible to thrombosis from infective emboli and may lead to the more frequent occurrence of chronic ulcer The acute ulcer may occur elsewhere throughout the stomach and duodenum but it heals more readily

In addition to peculiarities of the blood

supply the distribution of the lymphoid follicles may also play a part in the greater susceptibility of certain regions of the stomach and duodenum to the formation of ulcers Such follicles are much more common in the pyloric portion of the stomach than in the cardiac region They also are more frequently encountered on the lesser curvature than on the greater curvature and also predominate in the duodenal bulb An abscess developing in such a follicle may perhaps be a factor in the eventual origin of an ulcer as originally suggested by Cruveilhier

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Of the total number of gastric ulcers 9

were on the anterior surface, 27 on the posterior surface, 41 on the lesser curvature, and 19 on the greater curvature Eight were at or near the cardia and 15 others were within 8 cm of the cardia Of the total, about 76 per cent were near the pylorus, 12 per cent near the cardia, and 12 per cent in the midgastric zone

With regard to the factor of sex, 89 of the patients with gastric ulcer were males and 31 females This is approximately a ratio of 3 to 1 Thirty four of the patients with duodenal ulcer were males, 10 were females This again is a ratio of about 3 to 1

The greater frequency of gastric ulcer as compared with duodenal ulcer as demonstrated in our Bellevue Hospital autopsy records was substantiated also in the autopsy studies by Hart[6] and by Portis and Jaffe[7]

Such findings are contrary to surgical as well as roentgenologic findings Even an average experience with the roentgenology of peptic ulcer shows that duodenal ulcer preponderates considerably in frequency as compared with roentgen evidence of gastric ulcer The reason why duodenal ulcer appears to be much more common than gastric ulcer, radiologically and surgically, can perhaps be found in the greater tendency of acute gastric ulcers to heal spontaneously Also, some of these gastric lesions may be so superficial as to defy detection roentgenologically

ROENTGEN DIAGNOSIS

A revolutionary advance in the recognition of the presence of gastric ulcer resulted from the development of the roentgen method of examination In 1906 Hemmeter[8] published the results of his researches He produced ulcers in rabbits and cats experimentally by creating a defect in the gastric lining He removed a ring of tissue 1 cm in diameter, down to the circular layer of muscle he then coated this denuded surface with bismuth subnitrate and after closing the abdomen was able to visualize the region by means of the roentgen rays

Hemmeter then endeavored to demonstrate in a similar manner the presence of such experimentally produced denudations by introducing a bismuth suspension to spread over the lesion This could be done provided less than 48 hours had elapsed for he found that the base of the ulcerated area was rapidly covered with granulation tissue In a like manner he endeavored to visualize ulcers in human subjects by having the patient drink a suspension of bismuth in water on an empty stomach Owing to the fact that the chronic ulcer forms a crater, frequently covered by an agglutinating transudate, he believed that this would *mechanically cause the bismuth to adhere* and become demonstrable with the roentgen rays He succeeded in this in three cases One of the patients in whom an ulcer was diagnosed in this manner was operated on by Dr John B Deaver and the roentgenologic findings were confirmed

Reiche[9] in 1909 had occasion to examine, by means of roentgen rays, a patient, aged 47 who had a history of serious gastric disturbance of several years duration He noted in the middle of the lesser curvature of the bismuth filled stomach a small, mushroom shaped protuberance After 7½ hours the stomach was still almost entirely filled but it emptied at 20 hours At this time the projecting area was also free of bismuth The patient vomited coffee ground material and passed tarry stools At autopsy, an ulcer of the lesser curvature was found this was round, about the size of a quarter, deeply penetrating and had smooth margins The floor of the ulcer was formed by the pancreas and within it was a vascular lumen closed by a fresh thrombus The depth of the ulcer in the extirpated organ was not as great as one might have been led to believe from the size of the pouch in the roentgen picture

In this remarkable paper Reiche proved the following points of a fundamental character

1 That a practical method of diagnosing chronic gastric ulcer is by demonstrating a pouchlike projection of the lesser curvature

of the stomach seen in the roentgenogram This was verified with absolute certainty by autopsy

2 That although the ulcer was not present at the pylorus and no constriction was noted in that region a serious disturbance in gastric motility resulted giving rise to retention to a pathologic degree

3 That the actual size of the ulcer did not correspond to the apparent size as seen in the roentgenogram This Reiche explained by assuming that the increased intragastric pressure pushed out the nonresistant ulcer bearing area devoid of its muscular layers and thereby excluded from peristaltic activity

Haudek[10] in 1910 elaborated upon and popularized the roentgen method in the diagnosis of ulcer He verified its practical value in numerous cases that were proved to be correct by surgery or autopsy He summarized the roentgenologic evidence as follows (1) a diverticular like projection of the stomach generally of the lesser curvature (2) a gas bubble above it when the patient remained erect (3) a residue of bismuth in it visible for a protracted period (4) the inability to influence this area by palpation Of all the e however the first (the presence of a niche) is the only absolute diagnostic criterion

The Niche While the term niche historically refers to the roentgen appearance of the perforating type of ulcer it is at present used more universally to apply to the pocket produced by ulcers regardless of the degree of penetration or pathologic destruction of the layers of the stomach

The most important evidence of ulcer is obviously the niche which is the roentgen counterpart of the ulcer itself Indirect signs may occasionally be of value as evidence of a suggestive character The larger the number of indirect signs the greater is of course, the probability of a correct diagnosis However a diagnosis based on such evidence cannot be made with absolute certainty since these findings may be mimicked by disorders of a functional nature It is for this reason that every effort should be made to demonstrate the niche itself, which is the ultimate diagnostic criterion of ulcer

In examining the stomach for roentgen evidence of ulcer, it is important to remember that while such lesions are usually at or near the lesser curvature they may al o be on the posterior wall some distance away and almost anywhere in the stomach from the cardia to the pylorus

The important point regarding this fact is that the stomach must be examined through every angle of obliquity in order to determine whether the niche of an ulcer is actually present A patient may be examined with the rays directed postero anteriorly that is to say with the patient facing the fluoroscopic screen and no deformity of the lesser or greater curvatures of the stomach may be noted in this position However if this same patient is gradually turned so that his right side is placed against the screen (first oblique position) the niche may eventually appear as a projection of the lesser curvature border Sometimes the patient may have to be turned until his position is practically lateral to the screen before any such evidence of niche formation is brought clearly into view This means of course that the ulcer is present on the posterior wall of the stomach The patient should also be turned in the opposite direction so that the left side is against the screen (second oblique position) In some cases an ulcer on the posterior wall may then be noted as a projection of the greater curvature Moreover the change from the vertical to the prone position or vice versa may make a difference in the ability to visualize the niche

In addition to examination of the stomach through every degree of obliquity mucosal relief studies by the thin layer method may also be essential for the recognition of a lesion on the posterior wall so centrally located that it cannot be made to project from either the lesser or greater curvature borders of the stomach The mucosal technic in such cases may show the presence

of an ulcer otherwise completely obscured by the large amount of barium anterior to it It is for this reason that, in examining a stomach for a suspected lesion, under fluoroscopic control, the patient should be instructed to swallow only one mouthful of the barium suspension *at a time After* this is spread manually over the mucous membrane the lesion may be brought into focus In those cases in which special mucosal studies are desired the technic is *recommended which is outlined in the chapter* on the study of the normal gastric mucosa

As a rule, the niche is rounded and has a comparatively smooth outline In rare cases, the contour may be irregular, even though the lesion is benign as the result of the protrusion within it of a blood vessel or shortly after a subacute perforation when a thin stream of barium may be noted extending somewhat beyond the confines of the niche itself Barium within the niche may show an irregular distribution due to the character of the surface of the lesion resulting from various gradations of destruction and regeneration of the tissues within the ulcer itself Intrusion of pancreatic tissue within an ulcer that has perforated posteriorly may be responsible for the *appearance of rounded translucencies within* the confines of the niche

Of considerable practical importance is the distribution of various layers within the confines of the niche as seen in the *erect position Thus in some cases instead* of the niche being filled with a homogeneous mass of barium one may note the presence of three layers (1) a dense layer of barium at the lower pole of the niche surmounted by *(2) a less dense layer with a fluid level* which is (3) capped by an air bubble As a rule a niche with an appearance of this kind signifies that the ulcer is of the perforating type that is to say it represents an ulcer which has slowly perforated through all the coats of the stomach and is walled off by adhesions An ulcer like this will frequently be found adherent to the pancreas or the under surface of the liver It is fixed in position so that palpatory manipulation of the niche behind the fluoroscope will show an absence of or marked diminution of mobility Another important feature regarding a niche with *this three layer appearance is that, with* the rarest exceptions, it indicates that the ulcer corresponding to it is benign An exception to this rule will be found when the subject of malignant ulcer is discussed later on At that time also, the differential diagnosis between a benign and a malignant ulcer will be described

Complicating the problem of such differential diagnosis still further are a number of other features which may simulate the appearance of a niche, although ordinarily such differentiation should be a comparatively simple matter A diverticulum of the duodenojejunal junction may, in rare cases simulate the niche of a gastric ulcer when it overlaps the lesser curvature Careful fluoroscopic observation should make such differentiation readily possible By turning the patient through various degrees of obliquity, the diverticulum will be found to bear no relation to the lesser curvature of the stomach itself Careful inspection will also show that the diverticulum springs from *the duodenal curve It is only when one* attempts to make a diagnosis from the roentgen appearance on a single film that such a mistake may be made

Confusion of a niche with an atypical *peristaltic wave may also occur but only* when one attempts to make a diagnosis from a single roentgenogram Obviously, fluoroscopic observation showing the inconstancy of such a finding and its complete *obliteration by progressive peristaltic waves* will eliminate any such possible diagnostic error

At times a small amount of barium at the duodenojejunal junction as it emerges *behind the posterior wall, may also simu*late the niche of a gastric ulcer in the single roentgenogram Such an area may be recognized by the characteristic appearance

produced by the valvulae conniventes as well as its transient localization particularly as determined on fluoroscopic observation, and its incorporation within the rest of the duodenojejunal curve

A calcified area of extragastric origin in relation to the lesser curvature of the stomach, may also be differentiated from a niche by the fact that its shadow differs from that produced by barium it is completely separable from the stomach and the shadow is present either prior to the administration of the barium or long after the alimentary tract has been completely emptied

Differentiation of an ulcer from a gastric diverticulum will be discussed in more detail in the chapter devoted to a description of the latter abnormality The essential features in the differential diagnosis are

1 A gastric diverticulum as a rule is located on the posterior wall near the extreme cardiac end of the stomach to the left of the entrance to the esophagus Gastric ulcers that high in location are very rare

2 A *gastric diverticulum as a rule* has a neck extending from the stomach and communicating with the saccular dilatation A communicating neck of this nature is not ordinarily seen with ulcer

An important point in the roentgen demonstration of the ulcer is the possibility of determining its actual depth from the appearance of the corresponding niche In the case of a chronic ulcer the walls of which are rigid and in the resected specimen feel almost cartilaginous the size of the niche may correspond closely to the actual ulcer itself The condition is different however in the case of ulcers that have not been stiffened by considerable proliferation of connective tissue In such cases the ulcer in the resected specimen may show considerably less depth than the corresponding niche in the roentgenogram In order to explain *this apparent discrepancy the role* of the gastric mucosa must be taken into consideration During life with the ulcer intact there is a heaping up of the gastric mucosa in the wall surrounding the ulcer as a result of edema and spasm Roentgenologically, therefore the actual depth of the niche depends on two factors (1) the anatomic depth of the ulcer itself and (2) the exaggeration of this depth by the swollen mucosa In the resected specimen or at autopsy the increased prominence of the mucosa is not present and the depth noted is that of the actual ulcer itself

That discrepancy between the depth of the ulcer, as seen in the resected specimen and the exaggerated depth as observed roentgenologically is dependent upon the heaping up of the gastric mucosa surrounding the site of the ulcer is brought out by the work of Konjetzny [11] who treated the gastric mucosa in such a manner as to preserve the swelling of the folds and thereby demonstrated this to be the essential factor in the apparently increased depth of the niche as observed roentgenologically during life

As for the comparative depth of the niche in the roentgenogram and of the ulcer as seen *after resection it may actually be* identical provided the proper precautions are taken A common source of error in a comparative study of this nature lies in the fact that the resection of the ulcer may be undertaken quite some time after the original roentgen examination During this time a certain amount of healing may have taken place even though it is not sufficient to cause an amelioration of the symptoms If the ulcer in the resected specimen is then compared with the original roentgenogram considerable discrepancy in the comparative depth will be noted For the comparison to be accurate the roentgen examination should have been made as soon before operation as possible Moreover the appearance of the ulcer should be gauged immediately after resection Otherwise as the specimen gets older, it may undergo changes which *cannot then be accepted as* indicating the true depth of the lesion during life

A question often asked is whether one can determine from the roentgen appear

ance of the niche itself the extent to which an ulcer has involved the various coats of the stomach Without an understanding of the role the swollen mucosa surrounding the ulcer plays in exaggerating its depth, one might naively assume that a niche which in the roentgenogram appears to possess considerable depth necessarily means destruction of more tissue in the wall of the stomach than has occurred in the case of a niche that is less deep However, because of the variable nature of this extremely important factor of the heaping up of the folds of mucosa along the margin of the ulcer, no parallelism can be drawn between the depth of the niche and the degree of penetration through the layers of the gastric wall

With regard to the most superficial ulcers, roentgen examination may prove to be entirely negative or may present only indirect evidence such as an incisura localized tenderness and delayed gastric evacuation Careful mucosal studies, however may show even a superficial lesion by the demonstration of a niche This is particularly true because the heaping up of mucosal folds at the wall of the ulcer may exaggerate the apparent depth of the lesion A niche that is therefore readily recognizable roentgenologically may be caused by a lesion that is anatomically superficial in character This is important, since it not only makes roentgen examination of a superficial ulcer possible but also explains why at operation, the surgeon may fail to recognize such a lesion unless the stomach is opened Since the ulcer has not penetrated deeply enough to involve the serosa the surgeon may have considerable difficulty in locating it by external palpation alone

Roentgenologically the niche may appear as if completely separated from the stomach, because of the displacement of barium by the swollen mucosa surrounding the wall of the ulcer A knowledge of the pathologic physiology underlying this phenomenon makes us realize that such apparent separation of the niche from the contour of the stomach does not necessarily mean that we are dealing with a perforating ulcer Moreover, it enables us to understand one of the reasons why an ulcer may occasionally fail to be visualized roentgenologically For if the heaped up folds of mucosa surrounding the ulcer have caused complete occlusion, it may be impossible to force barium from the stomach into the ulcer itself

This may be considered as a protective mechanism It may possibly be a factor in diminishing the tendency toward perforation of an ulcer as a result of the increased intragastric pressure against it Gutzeit on the basis of gastroscopic observation, has shown that the swollen mucosa surrounding the wall of an ulcer may at times be approximated in so close a manner as to occlude the area of communication between the ulcer and the stomach With diminution in the edema and swelling of the surrounding mucosa the ulcer may then be recognizable both roentgenologically and gastroscopically

Other Evidence of Ulcer The following are among the indirect evidences of ulcer First there may be a localized permanent spastic incisura of the greater curvature Though sometimes seen in association with the niche of the ulcer, on the lesser curvature opposite, it may on occasion be the only evidence of abnormality Such an incisura, to be significant, should be permanent However the possibility of a functional origin must always be considered, although in this type the incisura is more apt to be inconstant than when associated with ulcer While such incisuras of ulcer ordinarily occupy the same plane as the ulcer itself, they may at times involve the greater curvature at some distance from the lesion The incisuras may vary in depth but as a rule are narrow and sharply defined

Of some importance in the roentgen recognition of ulcer in the absence of definite niche formation is the contribution of Frankel[1] who recommended the application of roentgen cinematography for the demonstration of ulcers of the stomach

without niche formation He took sixteen roentgenograms in a period of 20 seconds and by superimposing these, was in some cases able to demonstrate a small straight area on the lesser curvature which remained constant while the rest of the contour showed alterations produced by peristaltic activity He called this finding the ulkus riegel or crossbar which he interpreted as being due to a segmental suppression of peristalsis He applied the identical procedure to the demonstration of small areas of infiltration of the stomach which remained permanently unaltered while the rest of the contour was changed by peristaltic activity When associated with an incisura of the greater curvature opposite this local absence of peristalsis assumes even greater significance in the diagnosis of ulcer

I have noticed such localized absence of peristaltic activity in cases in which a niche had originally been present where the ulcer has apparently gone on to healing Possibly scar formation and contraction of the longitudinal muscle fibers may play a role in such cases Occasionally also I have observed a sharp smoothly outlined localized region of the lesser curvature without niche formation at the site of an ulcer However, with films taken through various angles of obliquity it has sometimes been possible to demonstrate the actual niche When the area has become stiff and superficially irregular the possibility of malignancy must be considered

Localized tenderness at various regions of the stomach is at best only suggestive and can never by itself be considered of great value

Of some interest in connection with the study of the appearance of the mucosal folds in relation to ulcer is the occasional evidence which may be roentgenologically demonstrable of radiating folds at the site of the ulcer bearing area

The radiation is apparently the result of three factors (1) contraction of the muscular layers at the site of the ulcer (2) eventual scarring in the case of the more chronic lesions and (3) according to Forssell swelling of the mucosal folds may be responsible as an additional factor

Prepyloric Ulcers While ulcers in the prepyloric area can frequently be readily diagnosed their recognition is not quite so simple as in the case of ulcers at or near the lesser curvature of the pars media Moreover a differential diagnosis between the benign and malignant nature of the lesion must be made with greater circumspection Holmes and Hampton[13] and Camp[14] have emphasized the seriousness of this problem although Singleton[15] found no essential difficulty in the roentgen differentiation of a benign from a malignant lesion in the prepyloric area in a series of cases subjected to careful histologic confirmation

The reasons for difficulty in the determination from a roentgen point of view of the benign or malignant nature of a lesion in the prepyloric region are the following (1) The detailed delineation of a small lesion in the pyloric region offers greater difficulties in this area than in the more proximal regions of the stomach This is particularly true if the pathologic process involves primarily the posterior wall (2) Lesions in the pyloric region are more apt to be associated with superimposed alterations of contour as a result of secondary spasm or chronic gastritis Such processes may produce annular narrowing of the pylorus irregularities and increased prominence of the mucosal folds and even translucent areas within the involved segment features differentiable with difficulty from those produced by a malignant process This danger of misinterpretation is heightened even more if the posteriorly placed benign lesion cannot be clearly brought into the foreground of observation because of technical difficulties (3) The problem of differential diagnosis in this region is complicated still more because of the fact that this is the area where changes produced by chronic gastritis as such may preponderate, where spasm of a reflex nature from an

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impression may be that of a stomach of which the distal portion has been altered by a malignant infiltration Prolonged, careful observation will finally demonstrate the true nature of the lesion

An important point in the roentgen diagnosis of hourglass stomach is the fact that the upper locule may at times so completely overhang the lower locule as to obscure the intermediary zone of narrowing Manual palpation with separation of the two locules under fluoroscopic observation may sometimes be essential in order to establish this diagnosis

For the hourglass biloculation of the stomach to be organic in origin it must obviously be a permanent characteristic of the viscus and remain unaltered in spite of repeated examinations or the administration of antispasmodics The stenosis producing the hourglass constriction secondary to ulcer is the result of three factors (1) spasm of the circular muscle fibers of the stomach (2) the presence of inflammatory edema and (3) actual organic stricture because of the formation of dense scar tissue

In the purely spastic type of hourglass stomach the incisura of the greater curvature is narrow and smooth in outline There may be variability in its exact appearance during prolonged fluoroscopic observation or on repeated examinations The spastic incisura may be in the same plane as an ulcer of the lesser curvature opposite which may be recognizable as a definite niche However spastic incisuras of the stomach may be of extragastric origin Owing to the spastic nature of the incisura there is rarely any obstruction to the flow of barium from the upper to the lower locule as noted in the stenotic type of narrowing produced by actual cicatrization of the organically produced hourglass stomach

It is an interesting clinical fact that in spite of the apparently bizarre and crippling nature of the deformity the presence of an organic hourglass appearance is not inconsistent with complete freedom from gastric symptoms Organic biloculation may be the result of an attempt on the part of nature to heal the primary ulcerating lesion and it may represent a residuum of this process

Rarely a cascade stomach may simulate the appearance of a true hourglass deformity In the cascade stomach however, the separation of the upper and the lower portions is not apt to be as sharply demarcated as in true hourglass stomach As a rule no associated evidence of organic pathology such as the niche of an ulcer on the lesser curvature is to be seen Variations in the position of the patient particularly in both the right and the left oblique views will show the typical 'cup and spill type of contour which is characteristic of the cascade stomach In addition intrinsic spasm apparently al o plays an important factor in this interesting phenomenon The inward curving along the greater curvature does not partake of the nature of an actual incisura and extends over a comparatively long area of the curvature

There are two other aids in differential diagnosis (1) by upward displacement of the barium the area of narrowing in the cascade stomach may be partly effaced and (2) if additional amounts of barium are ingested the stomach gradually becomes larger and a cascade deformity of the stomach may at times in this manner be made to disappear completely under direct fluoroscopic observation Occasionally on examination in the postero-anterior position the overhanging locule may extend beyond the confines of the rest of the lesser curvature in such manner as to simulate a gastric niche Examination of the patient in various positions however readily permits the true nature of the deformity to be ascertained

Another type of deformity which may simulate organic hourglass stomach is that produced by a scirrhous carcinoma of the midportion of the stomach There should rarely be any difficulty in differential diagnosis however In the first place malignant infiltration of the pars media is more apt to produce an irregularity of contour than

extragastric cause may develop and where benign tumors such as polyps may occur

For all these reasons, extreme care must be exercised in the determination of the nature of a lesion in this region First and *foremost is the value of the thin layer* method for the detailed visualization of the mucosal structure in this area An intact mucosa definitely favors the benign nature of the pathologic process The niche of an ulcer, if on the posterior wall, may then more clearly be brought into view and if it conforms in size and contour to the general features of the niche of a benign ulcer elsewhere *and there is no evidence of inva*sion of the surrounding mucosa, such a lesion may be considered as presumably benign In addition, alterations in the contour of the lesser and greater curvatures of the involved segment in various films would be in favor of a benign process Re exami*nation and comparative studies may show* sufficient alteration in the configuration of the involved area or diminution in the size of the niche when demonstrable, to justify an attitude of conservative watchful waiting as these would definitely indicate the benign nature of the lesion

As in the case of the niche of an ulcer elsewhere, it must be remembered that no criterion is absolutely final, and that in rare instances a lesion which seems to have features that would classify it as benign may eventually prove to be malignant However, with a careful evaluation of all the *details involved,* it should be possible to make a differential diagnosis in the large majority of cases between the benign or malignant nature of a prepyloric lesion to act as a guide in any therapeutic approach

Ulcer at the Pyloric Ring The following are the important changes which may occur at the pyloric ring as the result of ulcer in this region The pyloric ring may be angulated or slightly curved presumably as the result of scarring produced by the ulcer Another alteration may be that of elongation *of the pyloric* canal This may result from the narrowing of the immediate prepyloric region due to spasm scarring *or* possibly the exaggeration of the mucosal folds in this region The ring as a result of infiltration may become stiff and show no alteration in the size of the lumen throughout prolonged observation The most important evidence of an ulcer at the pyloric ring is the niche just as *it is the most reliable* criterion in the diagnosis of ulcer elsewhere

Deformities Resulting from Ulcer The presence of an ulcer may bring in its wake certain important anatomic changes which produce marked alterations in the size and shape of the stomach Moynihan[16] showed *that the conception of congenital hourglass* stomach was not tenable and that it was invariably secondary to organic disease One case he reported was that of a trilocular stomach due to old ulceration with cicatricial healing at two points 3 inches apart An excellent early monograph, from *a roentgen standpoint on the subject of* hourglass stomach is that of Rieder,[17] in which a large series of cases were subjected to surgical confirmation

Such abnormal configuration of the stomach may be of organic or of functional origin Even when the primary basis is *organic, there is an accentuation in the* degree of deformity by the added derangement of a functional nature In organic hourglass stomach, the narrow zone of communication is greater than in the spastic variety As a result, the two locules are separated by an elongated canal at the site of the lesser curvature Moreover, where the cause is organic due to scarring there is apt to be a superficial irregularity at the junction of both the upper and the lower locules A niche is not uncommonly *seen at the lesser curvature border* at the origin of the biloculation

As the area of communication becomes increasingly narrowed there is partial obstruction to the flow of barium from the upper into *the lower locule The barium* may then be noted passing slowly through this narrowed region and dropping to the lower pole of the stomach The primary

pyloric ring may in some cases be considerable I have on a number of occasions noted large residues in the stomach at 24 hours Surgical examination confirmed the fact that only a gastric ulcer was present

With ulcers at the pyloric ring there is a great tendency for occlusion with gastric retention This may be the result of either spasm or actual cicatrization and in many cases is probably due to a combination of both factors It is important to remember, however that spasm may be a significant factor in producing secondary gastric enlargement and abnormal retention This is particularly important to remember when a decision must be made as to whether medical or surgical management should be undertaken in a given case since a marked degree of gastric retention at 24 hours or longer is ordinarily considered a point in favor of surgical interference A knowledge therefore of the fact that spasm at the pyloric ring may be a complicating or indeed a predominating factor in pyloric obstruction is of considerable clinical and therapeutic significance The best means of differentiation is through an attempt at medical management including the use of antispasmodics and of gastric lavage The spastic factor may thereby be eliminated and the stomach may return to normal size and function A failure to alleviate the obstruction by this means would favor the diagnosis of actual cicatricial constriction of an unyielding character and might justify surgical intervention

Such pyloric stenosis however may also result from benign lesions other than ulcer as in cases of benign hypertrophy of the pyloric muscle with or without a coexistent ulcer and in some cases of carcinomatous infiltration An extragastric tumor may in rare instances produce sufficient occlusion of the pyloric lumen to simulate the stenosis produced by an intrinsic lesion

One of the earliest roentgen manifestations of pyloric stenosis is an exaggeration of peristaltic activity This may show itself in two ways (1) the number and depth of the waves may increase and (2) such waves may be seen originating high up in the cardiac end of the stomach It seems almost as if the stomach conscious of a difficulty to be overcome made a running start by beginning its peristaltic activity at the cardia Such increased functional activity may be able in the early stages to overcome the pyloric obstruction so that no abnormal retention results therefrom Gradually however, the stomach may become enlarged Evidence of gastric retention may develop The hyperactive peristalsis may alternate with periods of quiescence Finally periods of atony may become a prominent part of the picture and peristalsis may be superficial lacking vigor and ineffective in forcing the gastric contents through the pylorus This represents the stage of decompensation Barium retention may be present at 24 and 48 hours and even longer The stomach is considerably enlarged downward and particularly to the right

It is at this stage that the stomach at 6 hours may show the half moon appearance so characteristic of advanced pyloric stenosis This appearance is the result of a combination of two factors (1) the abnormal retention and (2) the fact that the marked enlargement to the right is as great as to the left It is important to realize that even at this stage spasm may be an important factor complicating and exaggerating the degree of pyloric stenosis Rarely, antiperistalsis may be noted

Because of the fact that the ingested barium in marked gastric enlargement may fall to the lower pole of the stomach upward displacement of the barium manually under fluoroscopic control may be essential to delineate the remaining contour Films in the prone position through various angles of obliquity are therefore important in order to demonstrate the integrity of the contour or reveal any evidence of irregularity which may aid in differential diagnosis This is particularly important since at times malignant infiltration of the pylorus

the hourglass deformity secondary to ulcer Secondly, carcinoma produces a dumbbell shaped deformity because of the annular narrowing of the midportion of the stomach. With ulcer the lesser curvature at the stenotic area is in line with the rest of the lesser curvature of the stomach, the deformity representing the appearance of the letter "B" Thirdly, the zone of communication in the organic hourglass stomach due to ulcer may be very narrow, even to the extent of producing some obstruction to the passage of barium through it With scirrhous infiltration of the pars media however, the channel is much wider and the barium traverses this region with considerable rapidity Owing to the infiltrative character of the lesion the capacity of the stomach in malignancy is considerably diminished Finally, in organic hourglass stomach due to ulcer the actual niche characteristic of ulcer may be demonstrable on the lesser curvature

Another important deformity of the stomach arising as a consequence of gastric ulcer is the snaillike retraction or "schneckenformige Einrollung" as originally described by Schmieden and Hartel [18] This is due to a shortening of the lesser curvature on either side of the niche as the result of the effect upon the longitudinal muscle fibers Both cicatricial contraction and spasm of the longitudinal muscle fibers play a part in this process Inflammatory changes in the lesser omentum originating in the ulcer may accentuate the process As a result of these various factors there is a close approximation of the proximal and distal portions of the stomach on either side of the ulcer The pylorus is thus pulled over to the left In some cases, the degree of retraction may be so extreme that the pyloric antrum and the duodenal bulb appear to be partially obscured by the body of the stomach Because of the organic nature of the process it is impossible in the advanced type of the deformity to separate the proximal and distal portions of the stomach manually under fluoroscopic observation The niche of the lesser curvature, while readily demonstrable in the early stages, may defy detection as the process advances

Effect of Gastric Ulcer on Gastric Motility Of significance from a roentgenologic standpoint is the effect of ulcer on gastric motility When the ulcer is at or near the pyloric ring it may inhibit motor function through direct involvement of the ring as the result of spasm or actual organic cicatricial stenosis with gradual gastric enlargement Retention within it may continue for days

When the ulcer is at some distance from the pylorus, the cause of pathologic gastric retention is not so readily explained Rarely retention may be found at 24 and 48 hours and even longer although no complicating pathology of the pyloric ring is discoverable at operation While pylorospasm may be present, perhaps a disturbance in the gastric gradient[19] may so paralyze motor function as to be an important associated factor in the production of this result According to Bauer, however extirpation of the Magenstrasse in dogs never produced prolonged retention phenomena [0]

Marked gastric delay in emptying with occasional 24 hour retention may be due to an actual extension of the inflammatory process from the ulcer to the pylorus [1] In other cases, the hypertrophic pyloric stenosis is at some distance from the ulcer of the stomach and no evidence of pathology other than the hypertrophy is to be noted In one case (noted by Stone and Ruggles), the ulcer of the pars media had actually undergone healing as determined roentgenologically and at operation but the pyloric narrowing had become even more marked Presumably once the initiating factor of pyloric hypertrophy has begun further exaggeration of the hypertrophic process may then continue of its own momentum

The disturbance in gastric motor function in the presence of a gastric ulcer without associated organic involvement of the

ever, as well as the eventual roentgenologic and clinical features of the case, proved the lesion to have been malignant

With only one possible exception, ulcers of the greater curvature have in my experience invariably proved to be malignant In the one case in which the lesion was conceivably benign roentgen examination revealed a characteristic niche projecting from the greater curvature at the junction of the pars pylorica and the pars media This finding was persistent fluoroscopically and roentgenographically After medical management with amelioration of the clinical symptoms further roentgen study showed no evidence of this original deformity There is therefore, no surgical corroboration of the diagnosis [28]



Fig 111 Gastric ulcer

Illustrative Cases J S male aged 65 The patient gave a 5 months history of epigastric pain gnawing in character made worse by eating He had several episodes of melena and lost 35 pounds since the onset of his illness There was a long history of pulmonary tuberculosis Gastric analysis showed a marked subacidity Stool examination was persistently negative for blood

Physical examination of the abdomen was essentially negative

A partial gastrectomy was done for a gastric ulcer

The pathologic diagnosis was gastric ulcer

may produce the picture of pyloric obstruction seen with ulcer Unless the entire contour of the stomach is demonstrable, differential diagnosis between the presence of a benign and a malignant lesion may be impossible Under such conditions *the only* diagnosis one may justifiably render on the basis of the roentgen findings is that of pyloric obstruction of organic origin For all practical purposes, a 24 hour gastric retention may be accepted as definite evidence of the presence of an organic lesion



Fig 110 Gastric ulcer

Ulcers of the Greater Curvature Though gastric ulcer shows a decided predilection for the lesser curvature and particularly for the anatomic pathway of the stomach known as the "Engpasse" or "*Magenstrasse*," occasionally an ulcer may occur elsewhere, such as on the posterior wall, but only with extreme rarity does it occur on the greater curvature Ulcerations of the greater curvature should always suggest *the probability of malignancy*

Glaessner and Finsterer reported a case of ulcer of the greater curvature

The patient was a man, forty three years of age, with a history of three years' duration He complained of violent pain to the left of the epigastrium, which was made worse about one hour after eating and occasionally awakened him at night He had had three gastric hemorrhages The entire epigastric area was tender Roentgen examination revealed a large ulcer on the greater curvature which retained the bismuth at twenty four hours when the stomach was entirely empty At operation, the greater curvature of the stomach was adherent to the spleen because of a callous ulcer which had penetrated into the lower lobe of the spleen *The ulcer was also firmly fixed* to the tail of the pancreas The ulcer was resected and the spleen was removed Histologic study of the ulcer revealed no evidence of malignancy The patient made an excellent recovery

Sutherland [3] in his report of two cases of ulcer of the greater curvature of the stomach, found only one of these lesions to be benign In this particular case the roentgenogram showed a large niche on the greater curvature of the pars media toward which the rugal folds converged This was verified at operation, the ulcer perforating against the sheath of the pancreas Blaine [4] recorded three cases of ulcer, one of which was *operated on and the lesion found* on the greater curvature

Additional cases of benign ulcer of the greater curvature confirmed by histologic study of the resected region were reported by Popovic [5] and de Luna and Astier [6]

The importance of microscopic confirmation in such cases is well shown by Sproull [27] In the case cited by him both the roentgenologic evidence and the gross anatomic characteristics of the resected lesion favored the diagnosis of *benign ulcer of the* greater curvature Histologic study, how

1 cm apart The larger stoma is oblong and measures about 1 5 by 0 5 cm and is the entrance to a round hemorrhagic smooth walled cavity about 1 5 cm in diameter The lip of the cavity is undermined This cavity contains a green pea The other stoma is round and has a diameter of 0 7 cm and is the opening for another smooth walled cavity with a fibrous appearance and measuring about 1 cm The tissues for 1 cm around both these areas are quite indurated and the serosa over them is fibrous The mucosa is absent in both cavities beginning again at their edges

The microscopic examination showed no evidence of malignancy

The diagnosis was chronic ulcers of the stomach

Roentgen examination (Fig 112) revealed a niche at the lesser curvature of the stomach in the immediate prepyloric region There was considerable shortening of the lesser curvature in this region with retraction of the pyloric ring The stomach was of considerably increased size The preoperative diagnosis was prepyloric ulcer with construction of the pyloric ring and gastric enlargement Note that, on pathologic examination the tissue about the ulcerations was found to be indurated and the serosa fibrous This is evidently the reason for the shortening of the lesser curvature in the region of the niche with the retraction and narrowing of the pyloric ring The second ulcer which was found on pathologic examination was not demonstrated in the roentgen study Perhaps this was due to the fact as discovered by the pathologist that a green pea was present in the crater which prevented the entrance of barium



Fig 113 Gastric ulcer with incisura of the greater curvature

The niche of a gastric ulcer may be associated with an indrawing of the greater curvature This may be narrow or quite wide A smooth narrow incisura is illustrated by the next case

J P male aged 64 About 2 months be

no evidence of malignancy either in the stomach or in a lymph node which was included *in the specimen*

The roentgen examination (Fig 110) showed a sharply defined niche *of the* lesser curvature, pars media The lesser curvature to either side was normal This deformity had the classic characteristics of a benign gastric ulcer The niche was well rounded It was located *at the lesser curvature, pars media*, and was free of any evidence of associated infil*tration*

lesser curvature, pars media The lesser curvature to either side was of *smooth contour* There was no evidence of *infiltration* The preoperative diagnosis was gastric ulcer

An ulcer may be present in the immediate prepyloric region

G H male, aged 44 Nine years previously the patient had suffered from abdominal discomfort, relieved by food and alkalies which was shown by roentgen examination to be due



Fig 112 Prepyloric ulcer

to a peptic ulcer He remained well until 1 week before his admission to the hospital when he had a return of pain On the evening of his admission, he was seized with severe epigastric pain associated with collapse The pain became generalized and radiated up into the chest Physical examination revealed boardlike rigidity and rebound tenderness

Operation revealed a perforation of a gastric ulcer at the lesser curvature about $1\frac{1}{2}$ inches proximal to the pylorus Eight months later he returned to the hospital stating that he had been well until 3 weeks before At this time a subtotal gastrectomy was done *for* a prepyloric ulcer

The report of the macroscopic examination was On the lesser curvature in the prepyloric region, there are two *ulcers located about*

A somewhat larger niche is illustrated by the next case

L P male aged 51 A subtotal gastrectomy was done on the basis of a well substantiated diagnosis of gastric ulcer The operative findings were as follows There was a deep ulcer on the lesser curvature of the stomach with a firm indurated area surrounding it which felt suspiciously like carcinoma There were no nodes in this area There was an active ulcer in the first portion of the duodenum with edema and scarring, and one small node in the gastrohepatic ligament

The pathologic diagnosis was chronic gastric ulcer

The roentgen examination (Fig 111) showed a somewhat rounded niche of the

ulcer crater on the lesser curvature just proximal to the incisura angularis measuring about 1½ cm in diameter There was no evidence of infiltration Gastric analysis showed normal acidity Examination of the gastric contents and of the stool was strongly positive for blood

At operation there was a large indurated ulcer of the lesser curvature which was adherent to the pancreas There were also some adhesions from the posterior wall of the stomach to the pancreas at the pyloric end A partial gastrectomy was done

The pathologic diagnosis was of a chronic gastric ulcer which had completely perforated the wall of the stomach

Roentgen examination (Fig 113) revealed a large niche of the lesser curvature pars media The lesser curvature to either side of the niche was of smooth outline and somewhat retracted There was a narrow incisura of the greater curvature opposite The appearance was that of a benign gastric ulcer as corroborated by microscopic examination of the resected specimen The incisura of the greater curvature was probably of functional origin Neither the gastroscopic examination the surgical exploration nor the pathologic examination of the stomach after removal showed any abnormality of the greater curvature which might explain the incisura



Fig 115 Gastric ulcer with wide incisura of the greater curvative



Fig 116 Organic hourglass secondary to gastric ulcer

A wide indrawing of the greater curvature is illustrated by the next case

K S aged 58 During the preceding 15 years the patient had had recurring episodes of right upper quadrant pain somewhat relieved by food The pain was worse at night The stools were tarry on two occasions There was moderate weight loss A cholecystectomy and an appendectomy a number of years before admission to the hospital had failed to give relief of the pain

Physical examination of the abdomen was essentially negative except for tenderness in the epigastrium and the right upper quadrant and the scar of the previous laparotomy

Operation revealed an ulcer high up on the posterior wall of the stomach near the lesser curvature which was removed In addition evidence of cirrhosis of the liver was found

The pathologic diagnosis was chronic ulcer of the stomach

Roentgenographic examination (Fig 114A) showed a classical niche of the pars media with a broad incisura of the greater curvature opposite The anatomic appearance of the resected ulcer is shown in Figure 114B

The next case also illustrates a broad incisura of the greater curvature opposite a lesser curvature ulcer

J V male aged 52 The patient stated that 5 years previously he had vomited a small amount of blood Thereafter he had recurrent attacks of blood free vomiting Dur

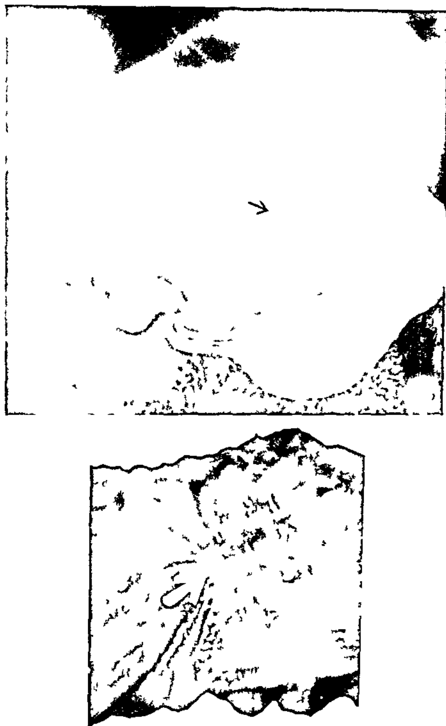


Fig 114 (A *Top*) Gastric ulcer with incisura of the greater curvature (B, *Bottom*) Same patient as is shown in (A) Anatomic appearance of the resected ulcer

fore his admission to the hospital, the patient was awakened from sleep by severe, generalized abdominal pain and the vomiting of dark brown fluid From then on he continued to have pain several times a day He had lost 30 pounds in the preceding 2 or 3 years Physical examination of the abdomen was essentially negative except for tenderness in the epigastrium

Gastroscopic examination showed a large

The pathologic diagnosis was chronic gastric ulcer

Autopsy 3 months after operation showed chronic fibrocaseous pulmonary tuberculosis

Examination of the remaining portion of the stomach showed it to be anastomosed to a loop of jejunum about 18 inches from the distal end of the duodenum, widely patent and well healed There was no evidence to indicate malignancy The liver was normal in appearance and there were no metastatic nodes

In spite of the report of malignancy of the stomach at the first operation the ulcer was proved ultimately to be benign The surgical *impression of the malignant nature of the* lesion may sometimes require histologic confirmation

From a study of the pathology at the site of the ulcer one may understand why the niche showed so much irregularity of contour The ulcer was adherent to the under surface of the liver and the floor had also penetrated to underlying tissue apparently pancreas and was so adherent that it could not be completely excised Inflammation edema and induration probably accounted for the wide incisura of the greater curvature in the region of the ulcer

Indrawing of the greater curvature the result of marked scarring may ultimately lead to an actual organic hourglass stomach

T L female aged 41 The patient had been complaining of recurring attacks of epigastric pain for a period of 13 years The pain was extremely severe radiated to the back and occurred immediately after meals On a careful diet she would improve for very brief periods Attacks of pain at night waking her from sleep occurred frequently She obtained no relief from food or soda She became gradually worse and lost 35 pounds in weight during the course of the illness She never vomited *until shortly before her admission to the hos*pital when he had a severe attack with coffee colored vomitus accompanied by the passage of tarry stools

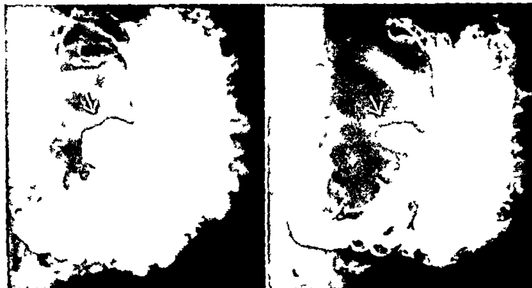


Fig 119 (A *Left*) Gastric ulcer Note the size of the niche when distended with barium (B *Right*) Same patient as is shown in (A) Note the diminished size of the niche with less barium

The physical examination was negative except for epigastric tenderness on deep palpation

At operation the stomach was found to be dilated attached and held up in the center in the shape of two separate pockets or bags due to an ulcer of the lesser curvature A posterior gastroenterostomy was done the stoma being attached to the proximal sac of the hourglass stomach

Roentgen examination (Fig 11b) showed evidence of an ulcer of the lesser curvature pars media A permanent indrawing of the

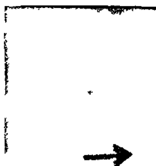


Fig 117 Gastric ulcer Note the translucent area within the niche apparently due to the intrusion of pancreatic tissue

ing the preceding year, he had vomited daily about 2 hours after meals and during the past 4 months, he complained of a dull epigastric pain lasting from 3 to 4 hours only occasionally relieved by food He had recently lost 20 pounds

Physical examination of the abdomen was essentially negative except for tenderness in the mid epigastrium The gastroscopic examination was reported as indicating a neoplasm of the posterior wall of the body of the stomach

Roentgen examination at that time showed the presence of an ulcer of the lesser curvature pars media of the stomach with a wide incisura of the greater curvature opposite The surgeon at operation reported that there was an irregular nodular indurated area of the lesser curvature of the stomach to which a small amount of omentum was adherent Small hard lymph nodes were found in the gastrohepatic omentum and scattered small nodules throughout the liver A diagnosis of inoperable carcinoma was made and the abdomen was closed without any operative procedure on the stomach itself No specimen was removed for biopsy

The patient s symptoms continued unabated, with progressive weight loss One year later the patient was readmitted to the hospital

Roentgen examination at this time (Fig 115) showed a large irregularly outlined niche of the lesser curvature pars media with a wide indrawing of the greater curvature opposite The mucosal folds appeared to be intact It was therefore believed that the ulcer was probably benign in spite of the fact that at the operation 1 year before, the surgeon considered the lesion to be malignant

The patient was operated upon again with the following description of the findings "Pathology Contrary to the previously recorded pathologic process, there was a tremendous crater defect on the posterior wall of the stomach penetrating into underlying tissue, supposedly pancreas There was a great deal of inflammatory reaction around the ulcer as evidenced by edema and induration Directly over this ulcer on the anterior wall was an indurated area in which there was some fibrosis to which the left lobe of the liver was adherent by means of an edematous band of tissue The liver was smooth throughout except for one portion in the left lobe which was white and scarred This was not interpreted as neoplastic change The stomach was not greatly dilated The pylorus was natural The greater omentum was adherent to the under surface of the transverse incisional scar"

A subtotal gastrectomy was done The ulcer could not be excised from the underlying tissue and the stomach was therefore separated from it by blunt dissection leaving the crater in the underlying tissue



Fig 118 Gastric ulcer showing an irregularly outlined niche with barium apparently leading from it

Roentgen examination (Fig 117) revealed a definite niche at the lesser curvature pars media At its apex was a rounded translucent area which was interpreted as having been produced by a blood vessel No such evidence was recorded by the surgeon In view of the fact that the ulcer had perforated through all the coats of the stomach with the pancreas forming the floor, it is possible that this translucent area at the apical portion of the niche may represent the actual intrusion of pancreatic tissue into the ulcer



Fig 121 (A *Left*) Note the cone shaped appearance of the niche (Examination made with patient in the erect position at 90°) (B *Right*) Same patient as is shown in (A) At this time the niche is well rounded The patient was in the erect right oblique position

Considerable irregularity in the contour of the niche may also be noted in Figure 115 in which connection the pathologic findings were described

Irregularity in the contour of the niche may be due to a sealed off subacute perforation as illustrated by the following case

J W male, aged 50 A few hours prior to his admission to the hospital, this patient had developed a sudden attack of severe pain in the left upper abdomen radiating up into the left side of the chest to the left shoulder and down the left arm On the evening prior to admission he had experienced paroxysmal dyspnea The sudden onset of pain on the morning of admission was associated with a general collapse The pain was aggravated by deep breathing Further questioning elicited the fact that he had been having left upper quadrant pain during the preceding 2 weeks

The physical examination was essentially negative The clinical suspicion was that of coronary occlusion

At operation there was found at the pars media on the posterior wall near the lesser curvature an area of induration with adhesions to overlying omentum The diameter was 3 cm A gastric ulcer was noted at this site A posterior gastroenterostomy was done with cauterization and suture of the gastric ulcer

Roentgen examination revealed an irregularly outlined niche with barium apparently

greater curvature separated the stomach into two locules, resulting in an organic "hour glass" appearance

Although the niche of a benign gastric ulcer is usually of smooth, well rounded contour when completely distended with barium, there are a number of factors which may influence its appearance One factor may be the intrusion of a blood vessel into the lumen of the ulcer When the



FIG 120 (A, *Left*) Rounded niche of a gastric ulcer (B, *Right*) Same patient as is shown in (A) With the *patient in the erect position, the niche appears cone shaped*

ulcer has perforated so that the floor is made up of pancreatic tissue, one or more translucent areas may be noted within the confines of the niche The cause of such a transformation is illustrated by the following case

M D, male, aged 52 Nine months before his admission to the hospital the patient was treated for gastric ulcer and felt well for 6 months when he had a recurrence of distress with vomiting which contained no blood The distress was located over the sternum and did not radiate

Physical examination was essentially negative, except for a right rectus scar at the site of an appendectomy

At operation a gastric ulcer was found on the posterior wall, which had perforated through all the coats so that the body of the pancreas formed the floor of the ulcer

A subtotal gastrectomy was done

Examination of the resected specimen revealed the following 'On the posterior surface at the lesser curvature 8 cm *proximal to the pylorus is an elliptical hole in the stomach* wall 2 5 cm x 4 cm in diameter Its edges are thickened The mucosal surface of the *specimen presents markedly hypertrophic* folds localized at the cardiac half which dwindle gradually to fine small rugae at the pylorus Microscopic examination reveals an ulcerated gastric wall with destruction of the epithelium, submucosa, muscularis and serosa There is a marked lymphocytic infiltration on the surface of which are strands of necrotic tissue and exudate The solitary lymph follicles are hyperplastic The adjacent mucosa is edematous and infiltrated with lymphocytes The mucosa adjacent to *the ulcerated area is* hyperplastic, but the muscularis mucosa in this area is intact" The diagnosis was gastric ulcer

in size The mucosal folds appeared to be unusually prominent The diagnosis of ulcer was corroborated by the operative findings

It is quite obvious that the size of a niche will vary with the quantity of barium in the stomach This is one reason why, in *studying the effect of any form of medical* therapy upon the size of the niche it is important that identical quantities of barium when the patient is erect may alter the appearance of the niche so that it may be cone shaped (Fig 121A erect posterior anterior position at an angle of ninety degrees) or rounded (Fig 121B erect first or right oblique position)

This is an important point to bear in mind in connection with the later discussion of alterations in the configuration of a

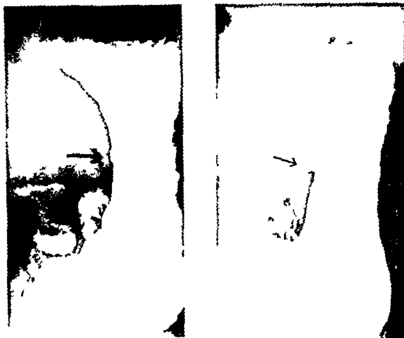


Fig 122 (C *Left*) Same patient as is shown in Figure 122 A and B With an increase in the degree of obliquity a small niche becomes visible (D *Right*) Same patient In an almost lateral position a large niche is demonstrable

be employed It is also clinically interesting that about 4 years previously the patient had been operated on for an acute perforation of a duodenal ulcer

The contour of a niche will vary with the position of the patient Thus a niche which is rounded in appearance when the patient is in the prone position (Fig 120A) may be cone shaped in the erect position (Fig 120B) This may be due in part to differences in the degree of distention of the ulcer with barium

Change in the degree of obliquity even niche in the healing process Every conceivable change in the position of the patient may cause concomitant variations in the appearance of the niche Moreover the niche of a gastric ulcer may completely escape recognition unless the patient is examined through every angle of obliquity This is illustrated by the following case

J F In Figure 122A this patient is erect *and the rays are directed at an angle of 90 to* him There is no evidence of the niche of a gastric ulcer Figure 122B shows the same patient turned through a moderate angle of

leading from it (Fig 118) This examination was made at the time of the patient's admission to the hospital The irregular outline of the niche and the apparent escape of barium from it were probably an indication of the imminence of perforation, which would correspond to the sudden onset of the left upper quadrant pain with collapse Roentgen examination 3 weeks later prior to operation showed a well rounded niche without irregularity or suspicion of escape of barium into the outside tissues The appearance of the niche in the original examination gave the impression of a subacute perforation which had become walled off

In some cases the only cause I have been able to discover to account for an intraluminal translucent area at the apex of the niche was a tab of omentum which had sealed off a perforation and apparently intruded within the confines of the ulcer

The appearance of a niche will vary with the quantity of barium within the ulcer as illustrated by the next case



FIG 122 (A *Left*) Gastric ulcer No niche is visible (B *Right*) Same patient as is shown in (A) First oblique position No niche is yet visible

P G, male, aged 45 This patient gave a 4 year history of upper abdominal distress from the time of his first admission to the hospital when he was operated on for a perforated ulcer of the duodenum From then on he was admitted on a number of occasions because of the recurrence of epigastric pain About 1 year after his first operation, he had a gastroenterostomy His most recent recurrence was of 1 month s duration it was characterized by epigastric pain radiating to the back and not related to food or alkali intake but relieved by alcohol He had occasional bloody vomitus and tarry stools While on the ward he had a sudden attack of severe pain in the upper abdomen which became diffuse

Physical examination of the abdomen revealed generalized tenderness and rigidity, most marked on the right side Operation revealed a perforated gastric ulcer at the lesser curvature

The day before perforation the patient had been roentgenographed Figure 119A showed a large niche of the lesser curvature of the stomach Figure 119B from a picture taken during the same examination but with less barium, showed the niche considerably smaller

The significance of the air-capped niche as evidence of chronic perforation is illustrated by the following case

E B, female, aged 60 The patient had been complaining for several years of pain coming on 1 hour after meals This pain was in the epigastrium and radiated toward the left side During the 3 weeks preceding her admission to the hospital the pain had been constant Occasional vomiting had occurred in diameter entirely ulcerated through the stomach wall and bound down by firm adhesions to the pancreas The thickness of the ulcer was 1 5 cm Microscopic examination of the ulcer showed no evidence of malignancy

Roentgen examination (Fig 124) showed a very large niche corresponding to the ulcer just described The fluid level and surmounting gas bubble were clearly shown That such a gas bubble was indicative of perforation was verified by the findings at autopsy, showing





Fig 124 (*Left*) Perforating gastric ulcer wi h an air bubble
Fig 125 (*Right*) Air capped niche with shortening of the lesser curvature

which did not contain blood She had lost 15 pounds during the preceding month

On physical examination marked tenderness was present in the midepigastrium One inch above and to the left of the umbilicus a smooth rounded mass was palpable

At operation, a very large firm indurated mass was pre ent the size of the palm of one s hand involving the midportion of the stomach There was a deep crater in its center No enlarged nodes were found Through the gastroenteric stoma the finger could be passed into a pouch extending into the lesser sac Resection was considered inadvisable and anterior gastroenterostomy was performed

At autopsy the stomach showed on its posterior wall a huge ulcer measuring 4 5 cm that all the coats of the stomach had been destroyed by the ulcer Note also that although the ulcer was large pathologic examination showed the lesion to be benign

Another factor of considerable importance is that of the shortening of the lesser curvature which in its extreme manifestation may produce a snail like type of stomach The following case illustrates this

J J male aged 38 This patient complained of upper abdominal pain for a period of several years The pain occurred from 15 to 20 minutes after food and radiated around the rib margins to the back There was periodic remission of the symptoms *He had lost 10*

obliquity There is still no evidence of any niche on the lesser curvature With the patient turned somewhat more obliquely, however one may perceive evidence of a niche of the lesser curvature, pars media (Fig 122C) The exact size of the niche, however, is not seen until the patient is turned in an almost lateral position (Fig 122D) An ulcer, therefore, is present on the posterior wall near the lesser curvature of the stomach This case illustrates the importance of examining a patient through every angle of obliquity in order to demonstrate the presence of an ulcer

An unusual factor in causing obscuration of a gastric niche is illustrated by the following case

M M, male aged 44 years This patient first noted epigastric pain about 1½ years before his admission to the hospital Since then the pain had been almost constant and varying in severity Two days prior to admission he had vomited coffee ground fluid He passed tarry stools that day The physical examination was essentially negative

The patient was operated on after preliminary medical treatment had failed to produce any amelioration of his symptoms At operation, an indurated ulcer was found on the lesser curvature at the junction of the pars pylorica and the pars media The crater was about the size of a dime A partial gastrectomy was performed Microscopic examination of the resected specimen failed to reveal any evidence of malignancy



Fig 123 (A *Left*) The niche of a gastric ulcer is clearly visible (B, *Right*) Same patient as is shown in Figure 123 A The niche is obscured by peristaltic waves

Of great interest is the roentgenographic evidence In the two accompanying roentgenograms both exposures were made with the patient in an identical position (erect) with the rays directed postero anteriorly Nevertheless, in Figure 123A the arrow points to a characteristic niche of the lesser curvature, corresponding to the ulcer found at operation In Figure 123B the arrow indicates the site where the niche should be seen but the niche cannot be visualized This is due to peristaltic activity the anterior portion of the stomach near the lesser curvature bulges and temporarily conceals the more posteriorly placed niche

J W male aged 55 The patient gave a 10 year history of recurring attacks of tarry stools and of vomiting of blood It was because of these recurrent episodes of bleeding that the patient entered the hospital for surgical therapy A partial gastrectomy was done

Pathologic examination revealed an ulcer of the lesser curvature about 5 cm proximal to the pylorus it measured 1 cm x 1 cm in diameter

Of considerable roentgenologic interest is the appearance of the mucosa in relation to ulcer In the first place its presence in association with a gastric niche is as a rule additional evidence of the benign nature of the lesion In some cases the folds are parallel and show no radiation to the site of the niche They may be unusually prominent either as the result of edema



Fig 127 Shortening of the lesser curvature of the stomach due to ulcer

Roentgen examination (Fig 127) revealed an area of marked shortening of the lesser curvature of the stomach at the junction of the pars pylorica and the pars media There was no demonstrable niche The evidence indicated the presence of an old cicatrized ulcer of the lesser curvature at the junction of the pars pylorica and the pars media

It is noteworthy that the last hemorrhage had occurred about 4 months before operation The roentgen examination was made 6 weeks prior to operation The ulcer in the interval between hemorrhage and roentgen study may very well have receded leaving the shortened lesser curvature as the major evidence of its presence

and spasm or at times as the result of an actual associated gastritis

An example of prominent mucosal folds in association with a gastric ulcer is illustrated by the case that follows

J C male aged 56 The patient gave an 8 year history of recurring attacks of epigastric pain after meals of vomiting and of hematemesis The pain was relieved by the ingestion of milk and by vomiting but at the time of his admission to the hospital it was constant Two days previously he had vomited about 1 pint of blood He had lost 40 pounds in weight Physical examination of the abdomen disclosed spasm throughout most marked

pounds during the preceding year In the 2 weeks before his admission to the hospital, diet failed to relieve the pain

Physical examination showed increased resistance over the right rectus muscle but was otherwise negative At operation, an abscess was found underneath the liver just to the right of the falciform ligament The entire stomach was adherent and bound down to the under surface of the liver and pancreas and great difficulty was encountered in delivering

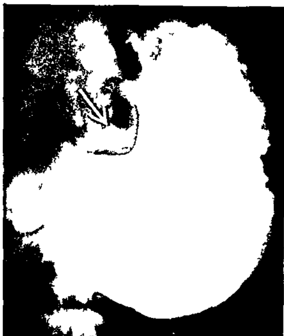


FIG 126 Marked shortening of the lesser curvature due to ulcer

the stomach into the operative wound Partial resection was done

Roentgen examination (Fig 125) showed a large niche on the lesser curvature with a fluid level and an air bubble In addition, there was marked shortening of the lesser curvature of the stomach particularly of that portion distal to the niche The pyloric arm was thereby drawn upward and toward the proximal portion of the stomach This shortening is due to the following factors (1) Spasm results in functional shortening of the lesser curvature at the site of the ulcer (2) Inflammation with the laying down of scar tissue and its subsequent contraction leads to secondary shortening of the lesser curvature (3) Possibly such a phenomenon may be exaggerated by the heaped up folds of mucosa at the site of the ulcer as the result of edema and inflammation When the shortening is of a permanent nature, one may reasonably assume that this is the result of actual cicatricial inflammatory change (4) In some cases, inflammatory involvement of the lesser omentum may cause shortening by an approximation of the lesser curvature both proximal and distal to the ulcer

In some cases, the only evidence of an ulcer may be the presence of shortening of the lesser curvature of the stomach No niche may be demonstrable This is illustrated by the following case

M S male, aged 47 For about 16 years this patient had been complaining of periodic attacks of epigastric pain these were not definitely related to meals He vomited blood and passed tarry stools He responded clinically to ulcer treatment The last episode began about 5 months before his hospitalization On the day of admission he had a sudden sharp abdominal pain accompanied by bloody vomitus and tarry stools

Physical examination of the abdomen revealed moderate abdominal distention with voluntary rigidity throughout There was moderate tenderness in the left upper quadrant and at the umbilicus

At operation the stomach was dilated and a markedly inflamed gastrohepatic omentum was found which was adherent to a large ulcer involving the lesser curvature, pars media causing constriction of the stomach The gall bladder and duodenum were normal The ulcer was thought to be too large and fixed to permit resection A gastroenterostomy was performed

The most significant finding on roentgen examination (Fig 126) was a persistent shortening of the lesser curvature at the pars media with an indrawing of the distal portion of the lesser curvature toward the proximal cardiac portion There was an incisura along the greater curvature in the immediate prepyloric region Apparently a factor in the shortening of the lesser curvature in this area was the markedly inflamed gastrohepatic omentum, which was adherent to the ulcer of the stomach and which contracted it in this region It is noteworthy that this patient showed not only a considerable gastric residue at 6 hours, but also a small amount of barium in the stomach at 24 hours

The significance of the shortening of the lesser curvature even in the absence of a visible niche is further illustrated by the next case

rated The ulcer was left in situ without dissecting it away from the pancreas The ulcer itself was closed A partial gastrectomy was done below the site of the ulcer because it was believed by the surgeon that a more radical resection would have endangered the life of the patient

Roentgen examination (Fig 132A) with the stomach completely filled with barium showed no evidence of any organic pathology The mucosal relief appearance (Fig 132B) showed a large rounded niche high up in the pars cardia corresponding to the ulcer on the posterior wall of the stomach found at operation



Fig 129 (*Left*) Radiating mucosal folds at the site of the niche
Fig 130 (*Center*) Radiating mucosal folds at the site of the niche
Fig 131 (*Right*) Prominent radiating mucosal folds at the site of the niche

It is interesting to note that even a large niche may be completely obscured when the stomach is filled with barium This case emphasizes the importance of mucosal relief studies in the demonstration of lesions that might otherwise be obscured

The importance of the thin layer method in showing an ulcer of the stomach which might otherwise not be visualized is illustrated by Figure 133A and B In Figure 133A the niche is barely visible In Figure 133B the niche is clearly visible

THE SIZE OF THE ULCER AND ITS HISTOPATHOLOGY

Mere size of the niche cannot be accepted as evidence indicating the probable malignant character of the lesion Assuming that all the other features in the roentgenogram point to the benign nature of the ulcer, a diagnosis that it is benign may be fully justified, even though the niche is of large size More important are the associated changes such as irregularity of the lesser curvature to either side of the niche and destruction of the normal mucosal pattern In the following case the very large niche was due to a benign ulcer

S H female aged 53 During the preceding 6 weeks the patient complained of severe pain which had begun in the lower abdomen but in a few days had become generalized finally during the preceding week becoming most marked in the umbilical region The pain was extreme and continuous with only rare periods of relief Vomiting occurred frequently shortly after eating The vomitus was usually dark brown in appearance and on one occasion was very black She had lost fifteen pounds during this period Physical examination of the abdomen revealed generalized tenderness and a sensation of a mass in the epigastric region

Operation revealed a large penetrating ulcer on the posterior wall of the stomach which had perforated and become adherent to the central portion of the pancreas The area of the pancreas about this point was hard and nodular

to the left of the epigastrium, where maximum tenderness was also present

At operation a gastric ulcer of the lesser curvature was found adherent to the pancreas The lesion was resected

The pathologic examination was chronic gastric ulcer

The roentgen examination (Fig 128) re



FIG 128 The gastric mucosa in relation to ulcer

vealed a classic niche of the lesser curvature pars media There was a fluid level within the niche capped by air The mucosal folds of the stomach were intact The evidence was that of a walled off perforating type of benign gastric ulcer

Some of the folds radiate toward the niche probably as a result of puckering of the mucosa Of considerable interest is the longitudinally placed translucent zone at the mouth of the niche representing the prominent wall surrounding the ulcer

Examples of the radiation of mucosal folds at the site of a niche are illustrated by Figures 129, 130 and 131

In the following case (Fig 131), the mucosal folds are shown radiating to the site of a large niche high up in the cardiac region of the stomach Note that the folds of the stomach appear quite prominent

E B., aged 46 At operation, high up on the posterior wall near the lesser curvature of the stomach, there was a huge ulcer plastered against the pancreas In addition, the scar of an old, healed duodenal ulcer was found Subtotal gastrectomy was done

The report of the macroscopic examination was as follows On opening the lumen the mucous membrane is found thrown up into high folds and well preserved except at the site of the defect high on the lesser curvature where the mucous membrane is perforated by an ulcer, the base of which appears to have been left behind' The microscopic examination revealed a benign gastric ulcer

The demonstration of an intact mucosal structure of the stomach is not only an important aid in establishing the fact that the ulcer is benign In some cases a mucosal study may be essential in demonstrating an ulcer which may otherwise escape detection This is particularly true when the ulcer is on the posterior wall Such was the situation in the case next to be described

G R, male, aged 58 The patient stated that 5 years previously he had had some stomach trouble which was diagnosed and treated as an ulcer He remained quite well until 2 weeks prior to his admission to the hospital when he had nonradiating, epigastric pain, occurring 2 hours after meals During the 4 days before admission, he vomited wine colored material and his stools were black He felt weak dizzy and short of breath He had lost 30 pounds in the preceding year

Physical examination revealed a white male, markedly dyspneic and extremely pale

Examination of the abdomen was essentially negative Operation revealed a large gastric ulcer on the posterior wall of the stomach high up on the pars cardia The ulcer penetrated into the tail of the pancreas The entire cardiac end of the stomach was indu

The pathologic diagnosis was gastric ulcer perforating into the pancreas

The roentgen examination (Fig 134) revealed a very large niche pars media The contour was quite smooth except near the upper pole The lesser curvature to either side of the niche was normal in appearance A *preoperative diagnosis of a benign ulcer* was made in spite of the size of the lesion This was based on three features (1) its location at the pars media (2) the essentially well rounded configuration and (3) the normal lesser curvature to either side of the niche which was free of any evidence of infiltration

An even larger niche of benign origin is illustrated by the next case

D W male aged 54 The patient was perfectly well until 3 months before his admission to the hospital when he developed heartburn and epigastric pain which was aggravated by food and relieved by soda The pain became more intense and radiated to the back He lost 29 pounds Gastric analysis showed a hyper acidity While on the ward of the hospital he had a tarry stool

Gastroscopic examination showed a large gastric ulcer which was reported as being probably malignant

At operation an ulcer approximately 2 5 *cm in diameter* was *found situated on* the posterior wall of the stomach near the lesser curvature pars media The ulcer was adherent



Fig 134 Large gastric ulcer benign



Fig 135 Large gastric ulcer benign

to the pancreas and was surrounded by considerable dense indurated tissue

The macroscopic examination was as follows The specimen comprises about seven eighths of the stomach On the outer surface at the pyloric portion of the lesser curvature the tissue is hemorrhagic and at the angle *there is a perforation 1 cm x 2 cm* The tissue here is stony hard On section this tissue appears as a ragged ulcer with necrotic walls and thrombosed blood vessels There are gray streaks of what appear to be tumor extending through the wall At the pyloric region there is again hard suspicious tissue

The microscopic examination of the ulcer and of a perigastric node revealed no evidence of malignancy The diagnosis was chronic gastric ulcer

The roentgen examination (Fig 135) showed a very large rather flat niche of the lesser curvature pars media It was superficially irregular The lesser curvature just distal to the niche appeared somewhat retracted but of smooth contour The preoperative *diagnosis was ulcerating lesion probably* malignant In this case in spite of the size of the lesion and the irregularity of its contour microscopic examination showed the ulcer to be benign

It is interesting to note that the gastroscopist thought that this ulcer was probably malignant and that the pathologist in the gross examination of the specimen de





FIG 132 (A, *Left*) Gastric ulcer on the posterior wall Note the failure of visualization with the stomach completely filled with barium (B *Right*) Same patient as is shown in (A) In the mucosal study the niche is now clearly visible





FIG 133 (A, *Left*) The niche is barely visible because it is obscured by overlying barium (B *Right*) In the mucosal study the niche is now clearly visible (Same patient as is shown in (A))

An example of two gastric ulcers in association with a duodenal ulcer is illustrated by the following case

C C male aged 62 The patient gave a 1 year history of epigastric pain relieved by food or powders There was no vomiting or bleeding Physical examination of the abdomen was negative except for slight tenderness to the left of the epigastrium

At operation a firm indurated lesion was felt high up on the lesser curvature of the stomach as well as an ulcer on the posterior wall of the duodenum A partial gastrectomy was done

Pathologic examination revealed two ulcers of the stomach There was no evidence of malignancy The duodenal ulcer found at operation by the surgeon had evidently not been included in the resected specimen

The roentgen examination (Fig 137) showed a niche of the larger ulcer high up on the lesser curvature of the stomach A smaller niche was present distally The duodenal bulb was deformed and there was a triangular shaped niche at the border of the lesser curvature The preoperative diagnosis on the basis of this evidence was two gastric ulcers and a duodenal ulcer The surgeon at the time of operation found evidence of only one gastric ulcer and the duodenal ulcer

Pathologic examination of the specimen when opened up along the greater curvature disclosed that two gastric ulcers were present

THE HEALING OF GASTRIC ULCER

That gastric ulcers frequently heal was shown by Brinton [9] many years ago He wrote The cicatrix by which the ulcer heals is on the whole about as frequently met with as the ulcer itself In other words half the instances of this disease undergo what is probably a spontaneous cure

That ulcers actually do heal is shown by statistical studies of autopsy material Thus Hirschfeld[30] reported that statistics in Vienna showed that 2 per cent of the patients examined at autopsy showed evidence of scars of ulcers that had undergone healing In Copenhagen the figure was considerably higher (20 per cent)

Stewart[31] also emphasized the fact that the healing of both gastric and duodenal ulcer is of common occurrence His observations revealed the fact that at autopsy healed scars were found with about half the frequency of active ulceration He gave conclusive histologic evidence of scars by showing fibrosis of the muscular coat With healing of the ulcer continuity of the mucosa is restored, although in such cases it is apt to be thinner and poorer in glandular structure The fibrous tissue underneath is apt to be less vascular Sturtevant and Shapiro in their statistical analysis of the autopsy material at Bellevue Hospital likewise showed that gastric ulcers not infrequently healed



Fig 137 Two ulcers of the stomach and a duodenal ulcer

Roentgenologically the niche may furnish us with an excellent criterion for determining changes in the ulcer Thus as a result of the healing process there may be a gradual diminution in size or even a complete disappearance of the niche There is also a life cycle in the history of a gastric niche so that it may come and go with periodic remission in the clinical manifestations of the disease When the niche remains unchanged in spite of treatment over a long period of time it is excellent evidence of chronicity and the unyielding nature of the lesion

Friedenwald and Baetjer[3] were the first to call attention to the value of roentgen rays in the study of the changes undergone

scribed the tissue as stony hard and stated that there were gray streaks of what appeared to be tumor extending through the wall and that there was hard, suspicious tissue in the pyloric region Clinically, also the brief history of 3 months of epigastric pain in a man of 54 with a loss of about 30 pounds in weight, would lead one to suspect a carcinoma Microscopic study, however, ultimately proved the lesion to be benign



FIG 136 (A *Left*) Large gastric ulcer benign Note the small air bubble at the upper *pole of the niche (B, Right) Same patient as is shown in (A) Note the appearance* of the wall surrounding the niche

The next case is that of a huge niche with evidence of chronic perforation

P C male aged 51 During the preceding year the patient complained of lower substernal and epigastric pain about 15 minutes after meals The pain radiated into the left chest to the back and on several occasions to the left shoulder There had been occasional vomiting without blood Nine years previously he had vomited about one half a cupful of blood The day before being hospitalized he had had a severe hemorrhage Physical examination of the abdomen was essentially negative

Operation revealed a large indurated mass on the lesser curvature adherent to the pancreas The lesion was resected Pathologic examination disclosed a large perforation of the stomach wall on the lesser curvature

The microscopic examination revealed chronic ulcer of the stomach

The roentgen examination (Fig 136A) revealed a very large, well circumscribed niche of the lesser curvature pars media The lesser curvature to either side of the niche was well defined and showed no evidence of infiltration At the upper pole of the niche was a small accumulation of air By means of the thin layer method the swollen wall of the ulcer could be demonstrated (Fig 136B) It was because of these findings that the diagnosis of a gastric ulcer was made, an ulcer which was *not only benign in nature but which had perforated* and become walled off

The presence of a fluid level capped by air at the upper pole of the niche seen upon examination in the erect position is usually an indication that the ulcer had previously perforated Moreover such an appearance is strong evidence in favor of the benign nature of the lesion I have *found only one exception to this rule*

Rarely more than one niche may be present in the stomach as a result of multiple ulcers

An example of two gastric ulcers in association with a duodenal ulcer is illustrated by the following case

C C male aged 62 The patient gave a 1 year history of epigastric pain relieved by food or powders There was no vomiting or bleeding Physical examination of the abdomen was negative except for slight tenderness to the left of the epigastrium

At operation, a firm indurated lesion was felt high up on the lesser curvature of the stomach as well as an ulcer on the posterior wall of the duodenum A partial gastrectomy was done

Pathologic examination revealed two ulcers of the stomach There was no evidence of malignancy The duodenal ulcer found at operation by the surgeon had evidently not been included in the resected specimen

The roentgen examination (Fig 137) showed a niche of the larger ulcer high up on the lesser curvature of the stomach A smaller niche was present distally The duodenal bulb was deformed and there was a triangular shaped niche at the border of the lesser curvature The preoperative diagnosis on the basis of this evidence was two gastric ulcers and a duodenal ulcer The surgeon at the time of operation found evidence of only one gastric ulcer and the duodenal ulcer

Pathologic examination of the specimen when opened up along the greater curvature disclosed that two gastric ulcers were present

THE HEALING OF GASTRIC ULCER

That gastric ulcers frequently heal was shown by Brinton [9] many years ago He wrote 'The cicatrix by which the ulcer heals is on the whole about as frequently met with as the ulcer itself In other words half the instances of this disease undergo what is probably a spontaneous cure

That ulcers actually do heal is shown by statistical studies of autopsy material Thus Hirschfeld[30] reported that statistics in Vienna showed that 2 per cent of the patients examined at autopsy showed evidence of scars of ulcers that had undergone healing In Copenhagen the figure was considerably higher (20 per cent)

Stewart[31] also emphasized the fact that the healing of both gastric and duodenal ulcer is of common occurrence His observations revealed the fact that at autopsy healed scars were found with about half the frequency of active ulceration He gave conclusive histologic evidence of scars by showing fibrosis of the muscular coat With healing of the ulcer continuity of the mucosa is restored although in such cases it is apt to be thinner and poorer in glandular structure The fibrous tissue underneath is apt to be less vascular Sturtevant and Shapiro in their statistical analysis of the autopsy material at Bellevue Hospital likewise showed that gastric ulcers not infrequently healed



Fig 137 Two ulcers of the stomach and a duodenal ulcer

Roentgenologically the niche may furnish us with an excellent criterion for determining changes in the ulcer Thus as a result of the healing process there may be a gradual diminution in size or even a complete disappearance of the niche There is also a life cycle in the history of a gastric niche, so that it may come and go with periodic remission in the clinical manifestations of the disease When the niche remains unchanged in spite of treatment over a long period of time it is excellent evidence of chronicity and the unyielding nature of the lesion

Friedenwald and Baetjer[3] were the first to call attention to the value of roentgen rays in the study of the changes undergone

by an ulcer under medical treatment They stated that they could thereby approximately determine the degree of healing of an ulcer which could not be as certainly determined in any other way

These observations were verified by the work of Ohnell[33] Hamburger,[34] Rosenthal[3] and White[36]

The value of the roentgen evidence of the healing of an ulcer is enhanced if repeated observations demonstrate a gradual diminution in the size of the niche over a period of weeks rather than an abrupt and complete disappearance of such evidence[37]

It is perhaps possible that the extremely rapid diminution in the size of the niche which is occasionally seen when the patient is undergoing treatment may not be entirely due to actual healing Another factor may be involved On the basis of the work of Forssell, a swelling of the folds of mucous membrane surrounding the active ulcer may be present as the result of hyperemia and the heaping up of irritated folds This would tend to make the niche appear to be much deeper than the corresponding ulcer actually was With rest in bed, a bland diet and antispasmodics, diminution in the swelling of the mucosa about the ulcer may result, so that the depth of the niche in the roentgenogram will agree more closely with that of the ulcer itself Such a change in the roentgenogram may be very striking, with out cicatrization of the ulcer itself necessarily having occurred



Fig 138 (A, *Left*) Niche of a gastric ulcer (B *Center*) Same patient as is shown in (A) The niche is smaller although the exposure was made during the same examination (C *Right*) Same patient as is shown in (A) and (B) The niche is still smaller here Figure 138 was obtained during the course of a single examination, with the patient in the same position

A recurrence of pain does not necessarily mean a recurrence of the original ulcer It may be due to the development of a new ulcer with new characteristics in the roentgenogram as to position and appearance However ulcers undergoing retrogression may fail to heal completely and after becoming considerably smaller may again show an increase in size

The evidence of ulcer healing as observed roentgenologically in the gradual disappearance of the niche is confirmed by gastroscopic observation Roentgenologically of course there are pitfalls which may lead to an incorrect conclusion regarding ulcer healing when this is based on the disappearance of the niche This is due to the fact that such failure to visualize a previously observed niche may result from occlusion of the ulcer with mucus a blood clot or food or from rapid diminution of the swelling of the mucosal fold surrounding the gastroscopist finds the healing process to be incomplete The average time for complete healing of an ulcer under effective treatment according to Schindler is 4½ weeks although in individual cases the duration may be more protracted Even enormous ulcers may heal with complete epithelization and no visible scar

An important point regarding the characteristics of the niche of a gastric ulcer is its contour Ordinarily rounded it may show irregularities at its base because of concomitant anatomic deformities An in

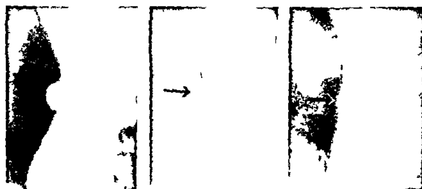


Fig 139 (A Left) Niche of a gastric ulcer (B Center) Same patient as is shown in (A) Note diminution in the size of the niche about 1 month after treatment (C Right) Same patient as is shown in (A) and (B) Almost complete disappearance of the niche about 1 month later A small cone shaped protrusion at the site of the original niche is still present

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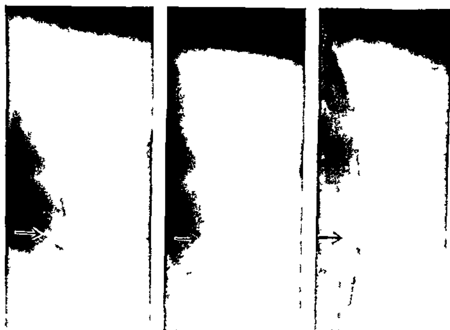


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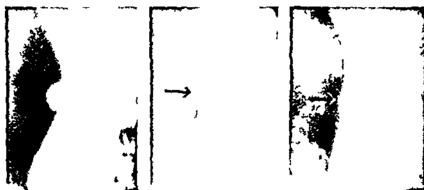


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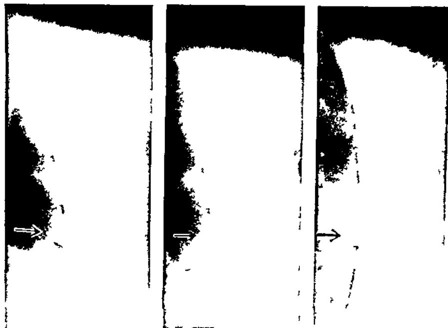


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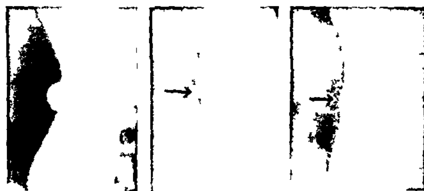


FIG 139 (A *Left*) Niche of a gastric ulcer (B *Center*) Same patient as is shown in (A) Note diminution in the size of the niche about 1 month after treatment (C *Right*) Same patient as is shown in (A) and (B) Almost complete disappearance of the niche about 1 month later A small cone shaped protrusion at the site of the original niche is still present

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a rigid unyielding wall, and (2) it favors a benign nature of the lesion, although here also exceptions to the rule have been reported

The niche of a gastric ulcer may also show an increase in size In some cases this may indicate the imminence of perforation (Berg) This however, is not an inevitable result of such enlargement That an ulcer may infrequently show an increase in size has been confirmed by the gastroscopic observations of Schindler

lesion, since obviously this could not have occurred in this case

That a niche may, however, become cone shaped in the healing process is well shown in the following case Figure 139A shows a moderate sized niche of the lesser curvature, pars media Figure 139B shows considerable diminution in the size of the niche It is cone shaped in appearance at this time Figure 139C shows almost a complete disappearance of evidence of the original niche A slight, triangular elevation at the



FIG 140 (A *Left*) Gastric ulcer (B *Center*) Same patient as is shown in (A), 20 days later Note the cone shaped appearance and the reduction in size (C *Right*) Same patient as is shown in (A) and (B) 2½ months later, showing complete disappearance of the niche

Illustrative Cases Evidence of moderate diminution in the size of a niche may occur even in the absence of healing This may be due to the mucosal folds surrounding an ulcer Transient changes in the degree of edema and swelling and spasm of the mucosal ring surrounding the ulcer may lead to concomitant changes in the apparent depth of a niche without actual healing having occurred This is illustrated in Figure 138A, B and C Note that the niche appears progressivly smaller in the three films which were taken during the course of a single examination with the patient in as nearly an identical position as possible Moderate diminution in the size of a niche, therefore does not necessarily mean that there has been an actual healing of the

site of the original niche is however, still to be noted

At times the niche may disappear so completely as to leave no trace of its original presence, as in the case that follows

E S, male aged 52 This patient gave a 1 year history of epigastric pain, definitely relieved by milk There was occasional vomiting the vomitus at one time containing a small blood clot Physical examination of the abdomen was essentially negative The Wasserman was 4 plus The patient did very well on medical management Paralleling the clinical improvement, there was corresponding evidence of the favorable affect upon the ulcer in the roentgen studies Figure 140A showed the size of the niche at the first examination Figure 140B, 20 days later showed a reduction in the size of the niche, which became tri

angular in shape Figure 140C, 2½ months later showed complete disappearance of the niche

At times in the healing process with disappearance of the niche, a sharply defined straight rigid area may remain this is unaltered by peristaltic activity and marks the site of the original niche The persistence of such an area of apparent stiffening may be regarded with suspicion since recurrence may take place at this site Such

'Proximal to and on the posterior aspect of the pars media of the stomach there was a large indurated area about 5 cm in diameter In the center of this area was a definite crater which admitted the tip of the little finger The pyloric ring was patent The ulcer bearing area seemed to be partially fixed to the *posterior abdominal* wall *No nodules were* palpable in the liver A gastroenterostomy was done

Roentgen examination about 5 months later (Fig 141B) showed the absence of any niche formation at the site of previous in

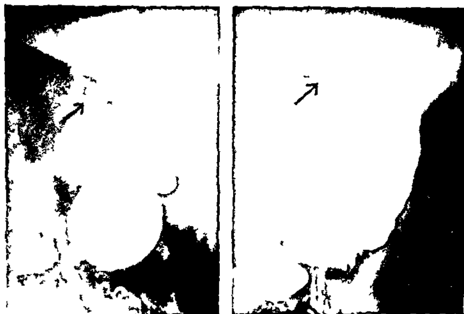


Fig 141 (A *Left*) Niche of a gastric ulcer (B *Right*) Same patient as is shown in (A) There is a rigid straight line at the site of the original niche 5 months after gastro enterostomy

recurrence of the ulcer took place in the following case

P B aged 52 The patient gave a 4 year history of epigastric pain occurring soon after meals and relieved by food There was occasional vomiting the vomitus was never *bloody or coffee* colored One month before admission to the hospital the patient noticed tarry stools Physical examination was essentially negative

Roentgen examination (Fig 141A) revealed a niche on the lesser curvature at the junction of the pars cardia and the pars media

The report after operation was as follows

volvement A rigid sharp bar was present at the site of the former ulcer Later there was a recurrence of the ulcer and eventually a partial gastrectomy was performed

The significance of a residual deformity in the healing process is further illustrated by the next case

T M male aged 57 The patient gave a 14 month history of epigastric pain radiating to the back occurring about 2 hours after meals and during the night and relieved by soda and food He vomited frequently There was no blood in the vomitus or stool He had lost considerable weight

Physical examination of the abdomen was

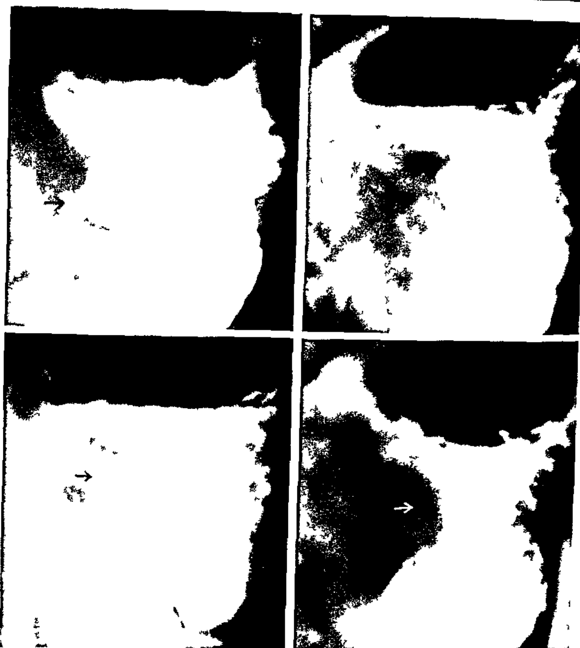


Fig 142 (A *Upper Left*) Large gastric ulcer (B *Upper Right*) Same patient as is shown in (A) Reduction in the size of the niche (C *Lower Left*) Same patient as is shown in (A) and (B) Still further reduction in the size of the niche (D, *Lower Right*) Same patient as is shown in (A), (B) and (C) Almost complete disappearance of the niche

negative except for epigastric tenderness He was treated medically with a satisfactory result clinically and radiologically

Roentgen examination (Fig 142A) showed a large niche of the lesser curvature at the junction of the pars cardia and the pars media Figure 142B showed a definite diminution in the size of the niche Figure 142C showed still further decrease in the size of the niche Figure 142D showed an almost complete disappearance of the niche There was however, a flat, cone shaped area marking the location of the ulcer and indicating that complete healing had not taken place in spite of the dramatic diminution in the size of the niche After remaining well for a

number of months the patient had a recurrence of intense epigastric pain and roentgen examination then showed that the niche had again become large At operation a large ulcer was resected which was high up on the lesser curvature of the stomach adherent to the pancreas The pathologic diagnosis was gastric ulcer

This case shows the gradual reduction in the size of the niche with almost complete disappearance but ultimately with a flare up in the symptoms associated with a marked increase in the size of the niche At operation a large ulcer was found

The life cycle of the niche of a gastric ulcer is demonstrated by the following

worse symptomatically Operation finally verified the diagnosis of gastric ulcer

In the following case there was first a reduction in the size of the niche followed by an increase in size, so that it ultimately was larger than in the original examination

R D male, aged 26 The patient gave a 6 year history of recurrent attacks of crampy pain in the epigastrium occurring about 1 hour after meals and not relieved by food or alkalies There had been frequent vomiting but no blood The pain sometimes awoke him from sleep Physical examination of the abdomen was essentially negative The patient was finally operated on and an ulcer about the size of a dime was found on the lesser

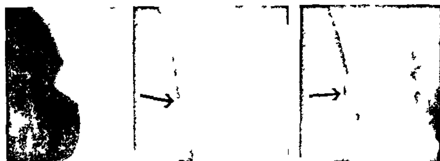


FIG 143 (A *Left*) Niche of a gastric ulcer (B *Center*) Same patient as is shown in (A) Disappearance of the niche 6 months later (C *Right*) Same patient as is shown in (A) and (B) Recurrence of the niche 4 months later

figure Figure 143A shows the presence of a definite niche of a gastric ulcer at the time of clinical activity of the lesion Figure 143B shows the absence of any niche formation 6 months later at a time when the patient was clinically well Figure 143C shows a recurrence of the niche at the location of the original lesion 4 months later which was coincidental with a recurrence of symptoms

An increase in the size of the niche of a gastric ulcer coincident with an increase in clinical symptoms is illustrated in the following figures Figure 144A shows the original appearance of the gastric niche Figure 144B shows the appearance of the niche 1 year later with marked increase in size The patient had become considerably

curvature of the stomach about 1½ inches from the pylorus The ulcer had penetrated all the walls of the stomach and was only closed by a thin membrane and a small portion of the lesser omentum A subtotal gastrectomy was done

The report of the pathologic examination was as follows Specimen consists of an ulcer about 1 cm x 1½ cm in diameter surrounded by mucosa varying from 1 cm to 3 cm in width The whole resected specimen is elliptical in shape The ulcer is about 1½ cm in depth and there is an opening through to the under surface The ulcer surface is somewhat hemorrhagic and in the subserosal area there is a blood clot and a portion of the omentum appears to be adherent to the area of perforation The rim of the ulcer is firm but not stony hard No lymph node is included

The microscopic diagnosis was chronic benign ulcer of the stomach

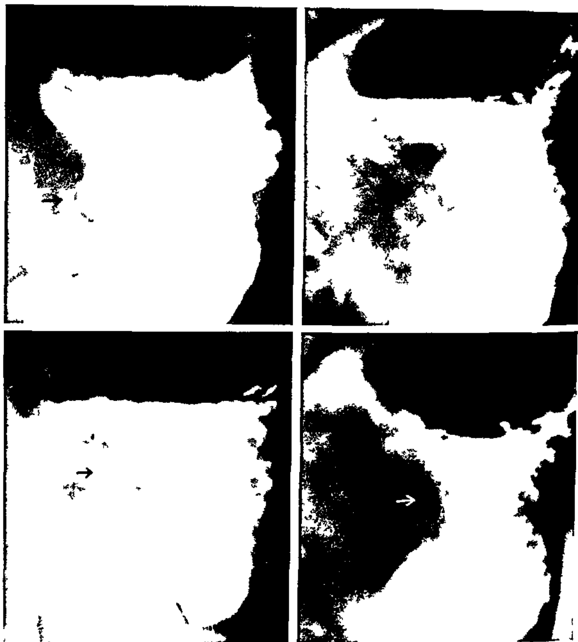


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In the following case there was first a reduction in the size of the niche followed by an increase in size so that it ultimately was larger than in the original examination

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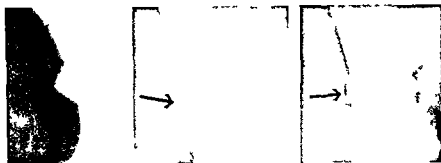


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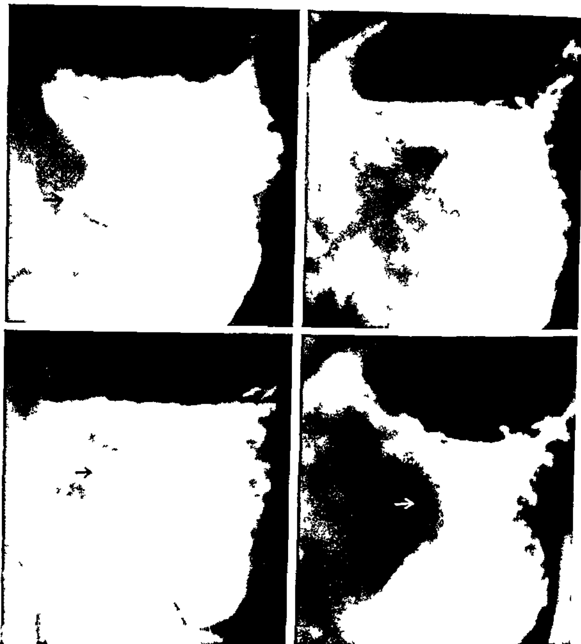


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destroy the cancer cells and leave only an apparently benign peptic ulcer in its place Such a process might explain the presence of a lesion of this nature in association with metastatic invasion and need not necessarily imply malignant change in a primary ulcer

Malignant transformation of an originally benign gastric ulcer is probably rare and the unusually high figures reported by Wilson and MacCarty [41] who stated that

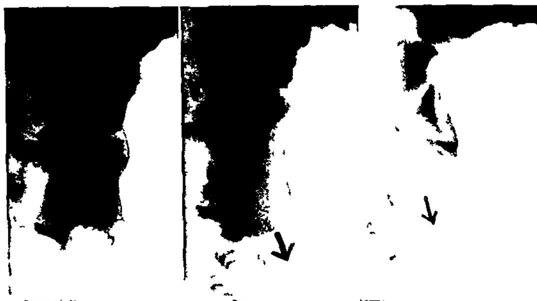


FIG 145 (A *Left*) Niche of a gastric ulcer (B *Center*) Same patient as is shown in (A) Three days later Note the diminution in the size of the niche (C *Right*) Same patient as is shown in (A) and (B) 7 months later Note the marked increase in the size of the niche

68 per cent of ulcer are complicated by carcinoma has been seriously criticized by Ewing [4] who regarded carcinomatous transformation of a peptic ulcer as infrequent and as being probably less than the incidence of 5 per cent established by many pathologists if only those cases are included in which there has been a long history of gastric ulcer where the tumor is limited to isolated foci or only one portion of the ulcer and in which the base of the lesion is free of malignant infiltration

THE ROENTGEN DIAGNOSIS OF MALIGNANT GASTRIC ULCER

As a rule the differential diagnosis of a benign from a malignant ulcer of the stomach should offer no difficulty When the ulceration is malignant a number of features help in its recognition These are discussed in the following paragraphs

1 In the presence of an ulcer of malignant character there is apt to be an invasion of the nearby tissues so that roentgenologically one may be able to demonstrate the presence of associated irregularities of contour of the neighboring wall with an absence of peristaltic activity in this area Moreover the diagnosis is simplified still further if there are irregularities of contour at some distance from the niche When the ulcer is benign the lesser curvature to either side is intact and shows continual alteration of contour due to the persistence of peristaltic activity When the mucosal structure of the stomach

Roentgen examination shows interesting changes in the appearance of the niche At the time of the first examination (Fig 145A) there was a small niche of the lesser curvature at the junction of the pars pylorica and the pars media Three days later (Fig 145B) examination showed a diminution in the size of the niche At this time, it was cone shaped Figure 145C, 7 months later, showed considerable increase in the size of the niche as compared with the appearance in the preceding examination, and it was also much larger than in the original examination (Fig 145A) The niche was sharply outlined and the lesser curvature to either side was smooth, the entire appearance being that of a benign ulcer The gradual increase in size was apparently due to a perforation of all the layers of the wall of the stomach As noted at operation and in the pathologic examination the perforation was walled off by a piece of omentum On further study of the niche a slight indentation near the inferior border was noted, *and the possibility suggested itself* with a knowledge of the operative findings in mind that this might represent the area of perforation sealed off by omentum

Multiple ulcers of the stomach may undergo simultaneous regression, as noted in the following case

G S male, aged 62 This patient gave an ulcer history and roentgen examination disclosed two ulcers of the lesser curvature (Fig 146A)

Figure 146B, about 2 weeks later, showed a reduction in the size of both niches Finally, all radiologic evidence of the ulcers disappeared although the patient still complained of some epigastric distress



Fig 144 (A *Left*) Niche of a gastric ulcer (B *Right*) Same patient as is shown in (A) The niche shows a considerable increase in size

THE RELATION OF BENIGN TO MALIGNANT GASTRIC ULCER

Apparently the first intimation that carcinoma might arise upon the basis of a primary *benign ulcer* was by Cruveilhier[38] and Rokitansky[39] both of whom, however, regarded the occurrence as rare Another important conception was that of Brinton,[40] who though he considered such transformation by no means rare, believed that it was possible for a new growth to become secondarily ulcerated in such manner as to

plete and permanent disappearance of the radiologic evidence

Rigler[43] in 1935 described a case in which carcinomatous tissue grew into the base of the ulcer simulating healing and another case in which the ulcer disappeared completely, although the carcinoma was increasing in size In this connection, the report by Palmer[44] is extremely interesting He included in his paper the histologic appearance of a scar adjacent to a malignant ulcer of the stomach This scar had become completely re epithelized with mucosa containing nests of tumor cells There was carcinomatous infiltration of the epithelial layer with neoplastic glands beneath the muscularis mucosae The major lesion however the malignant ulcer remained open and showed no attempt at epithelization

Perhaps the clearest example of such roentgen evidence of the disappearance of a niche of a malignant ulcer is portrayed by Schindler and Gold[45] The roentgenogram in the first observation showed a niche of the lesser curvature having the roentgen appearance of a benign lesion In the next observation there was a marked diminution in size and finally there was apparently complete disappearance of every vestige of the original lesion Autopsy revealed a diffusely infiltrating carcinoma of the entire stomach with widespread metastases An ulcer of the lesser curvature was fixed to the pancreas The surface of the ulcer was lined by tumor tissue and the base showed extensive fibrosis The accompanying photomicrograph of a section through the ulcer showed that not only had the floor of the ulcer become infiltrated with carcinoma but the gastric mucosa as well



Fig 147 Malignant gastric ulcer Note the rigidity of the lesser curvature particularly in the region distal to the niche

Mallory[46] stated that in one case in which roentgen examination showed a diminution in the size of the niche, patho-

is intact, this is in favor of the lesion being benign

2 The niche of a malignant ulcer may be irregular in outline and at times of unusual size However, neither the contour nor the size are reliable criteria the extent of ulceration may be considerable and yet the process may be benign

3 When the niche, even though very large, has a fluid level capped by air (in the erect position), one may consider the lesion as benign Only with the rarest exceptions has an ulcer having this appearance ever been encountered that later proved to be malignant

4 Another aid in differentiating a benign from a malignant lesion is the therapeutic test After medical management has been instituted, repeated roentgen observations may be made A diminution in the size of the niche, and particularly its complete disappearance, definitely favors the opinion that the ulcer is benign, although in rare instances this sign cannot be accepted as an absolute criterion If however, there is no evidence of a residuum of the niche no matter how small and if peristalsis through this area is normal, one may feel quite safe in eliminating malignancy as a cause of the original niche This does not, of course mean that if a niche fails to undergo retrogressive change under medical management it is necessarily malignant The reason for the failure to respond to conservative treatment may be the serious inflammatory pathology of the ulcer and the hard, unyielding character of its wall Also, a small cone shaped deformity of the lesser curvature of the stomach as a persistent residuum in the healing process may simply mean that the ulcer, even though benign, has not undergone complete restitution and that the integrity of the wall has not been reestablished Similarly, a straight rigid line may mark the site of the original niche and may indicate only that a weakness in the wall is present although the primary ulcerating process was benign



Fig 146 (A *Left*) Two ulcers of the stomach (B *Right*) Same patient as is shown in (A) Two weeks later Note the diminution in the size of the two niches

In rare instances the niche of an ulcerating lesion that ultimately proves to be malignant may apparently undergo retrogressive changes even to the point of com

of the lesser curvature as resembling a meniscus

6 A study of the gastric mucosa in malignant ulceration may show definite evidence of associated infiltration with destruction of the normal gastric relief and evidence of finger printing deformity

7 The location of the ulcer may be helpful in differential diagnosis since ulcers on the greater curvature are rarely benign

8 The combination of a malignant gastric ulcer and a duodenal ulcer is very rare Thus the presence of a duodenal ulcer would be a point in favor of the probably benign nature of an associated gastric ulcer

All these aids help materially in differentiating the niche of a benign from that of a malignant ulceration In rare cases however a niche which seems to meet the criteria that would ordinarily stamp it as belonging in the category of lesions that are benign is found on histologic study of the resected specimen to have been produced by a malignant process This possibility is however on the whole so small that it should not deter us from instituting conservative medical management in the treatment of gastric ulcer as recognized on the basis of careful roentgen observation

The problem of the benign ulcer origin of gastric malignancy from the radiologic point of view is complicated by the following factors First a roentgen diagnosis of benign ulcer is made yet at operation malignancy is found The original diagnosis may have been faulty because of the lack of a complete study of the lesion in all its details together with a careful roentgenologic study of alterations of the gastric mucosa or it may be an expression of the technical limitations of the roentgen method even in the hands of the astute and skillfully trained It certainly does not warrant the conclusion that the original lesion was necessarily benign and that carcinomatous degeneration occurred Secondly in a case in which a diagnosis of benign ulcer has been made the patient may live for several years and operative intervention may then demonstrate malignancy This also does not prove the ulcer origin of the growth, since the presence of malignancy is not incompatible with a number of years of life The original diagnosis may still have been misleading Thirdly, pathologic criteria of what constitutes early malignancy in any given lesion may not always be absolutely rigid and what one authority considers sufficient evidence for the diagnosis of cancer may not be acceptable to others In addition examination of such specimens may make it impossible to differentiate clearly between an appearance due to a malignant lesion that has undergone ulceration and an ulcer that has become malignant



FIG 149 Malignant gastric ulcer Note the rigidity and irregularity of the lesser curvature of the stomach distal to the niche

Another point against the ulcer origin of malignancy is the fact that a duodenal ulcer rarely if ever becomes cancerous I am unable to find a single case of this nature in the records of Bellevue Hospital Nor have I ever observed a case of malignancy in a postoperative jejunal ulcer

Schindler has never seen a benign ulcer transformed into a malignant one during gastroscopic observation over a period of years On the other hand ulcers which appeared benign roentgenologically and

logic examination of the lesion showed the former ulcer crater to be completely filled with carcinoma in situ

Similarly Eusterman showed the waxing and waning of a gastric niche as well as the disappearance of the ulcer and its recurrence under *gastroscopic control, only to* find at operation that the lesion was an ulcerating adenocarcinoma [47]

It is hardly necessary to emphasize the *fact before concluding that evidence of an* peared It must also be remembered that food or mucoid material or a blood clot lodged in the crater *may prevent the en*trance of barium for its visualization, and re examination may be necessary particularly if there is the slightest suspicion about *the findings and if they do not dovetail with* the clinical manifestations It is very probable that such a careful procedure in the corroborative examination of an ulcer patient will show that the complete disappear



Fig 148 (A, *Left*) Malignant gastric ulcer Note that the lesser curvature to either side of the niche is indrawn and rigid (B *Right*) Appearance of the resected specimen (Same patient as is shown in (A))

ulcerating lesion of the stomach has disappeared that an exacting technic is essential and that re examination should be made under the identical conditions employed when the lesion was first demonstrated This applies not only to the position of the patient relative to the ray but also, as nearly as possible, to the quantity of barium with which the stomach is *filled*

Mucosal studies and graded compression must also be an essential part of the technic before it is assumed that all radiologic evidence *of the original* lesion has disappearance of every roentgen vestige of a lesion of malignant nature is extremely rare indeed and for practical purposes, does not vitiate the clinical significance of such evidence as an indication of the benign nature of the original lesion

5 The niche of a malignant ulcer due to a crater within a carcinoma may appear as an indefinitely delimited shadow within a filling defect In the case of a benign ulcer the niche extends beyond the confines of the gastric contour Carman[48] emphasized the appearance of the crater of an ulcerating malignant lesion in the vertical portion

The significance of rigidity of the lesser curvature of the stomach to either side of the niche is further illustrated by the next case

Y H female aged 52 The patient complained of progressive weakness for a period of 2 years a weight loss of 34 pounds during the preceding year and epigastric pain after meals On physical examination, the liver was palpable but abdominal examination was otherwise negative Gastric analysis showed normal acid values At operation the surgeon stated that he found a large inflammatory lesion at the lesser curvature of the stomach A partial gastrectomy was done

H J female aged 40 The patient gave a 1 year history of occasional vomiting weight loss of 40 pounds and epigastric soreness There was no blood in the vomitus or stool Physical examination of the abdomen was negative Gastric analysis revealed a hyperacidity

Operation revealed an ulcer about 2 inches proximal to the pylorus which almost completely encircled the stomach It measured nearly 1 inch in thickness in some places The surgeon did not consider the lesion to be malignant

The pathologic examination was carcinoma of the stomach with secondary carcinoma in a lymph node



FIG 151 (A *Left*) Malignant gastric ulcer The niche is not clearly seen (B, *Right*) Same patient as is shown in (A) Mucosal study showing an irregularly outlined niche at the lesser curvature projecting within the lumen of the stomach Note the distortion of the mucosal pattern

The pathologic diagnosis was ulcerative adenocarcinoma of the stomach

Roentgen examination (Fig 148A) revealed evidence of a niche of the lesser curvature at the junction of the pars pylorica and the pars media The lesser curvature, particularly that part proximal to the niche was indrawn and rigid The impression was that of an ulcerating lesion that had produced infiltration and stiffening of the lesser curvature proximal to it

It was because of these findings that the ulcerating lesion was believed to be malignant Figure 148B showed the gross appearance of the resected ulcer

In the following case there was considerable distortion of the lesser curvature distal to the niche

Roentgen examination (Fig 149) showed a large niche of the lesser curvature, pars media The lesser curvature distal to the niche was indrawn and rigid in appearance and did not distend in a rounded manner similar to the greater curvature opposite The appearance was that of infiltration of the wall These findings pointed to the malignant nature of the ulcerating lesion As noted the diagnosis was confirmed by the pathologist although the surgeon at operation did not consider the lesion to be malignant

In addition to rigidity of the lesser curvature the fact that the niche does not project beyond it (as in the case of the typical niche already described) but remains within its confines is suggestive of ulcerat ve

even showed diminution in the size of the niche under therapeutic management were *found to be malignant from the start by* gastroscopic study Thus, the eventual *demonstration of the malignancy of a lesion* of this nature is no proof of the transfor*mation of a benign into a malignant ulcer*

Illustrative Cases The following is an *example of a malignant ulcer*

J K male, aged 60 The patient gave a 1 month history of pain in the epigastrium and in both upper quadrants The pain was sharp



Fig 150 (A *Left*) Malignant gastric ulcer The niche is not definitely visible *(B, Right) Same patient as is shown in (A)* Mucosal relief appearance showing irregularly outlined ovoid shaped niche, separated from the rest *of the stomach by a wide irregular translucent zone*

in *character and worse after food intake* Occasionally the pain radiated to the back He had had tarry stools during the preceding week and had lost 25 pounds since the onset

Physical examination of the abdomen revealed *the sensation of a mass in the left* upper quadrant At operation there was a large, fungating carcinoma of the distal third of the stomach There were numerous palpable nodes along the lesser curvature and several along *the greater curvature There* was a solitary node palpable in the liver

The report of the macroscopic examination was Specimen consists of the distal portion of the stomach unopened and unfixed measuring *about 15 cm in* length A large hard mass is palpable on the lesser curvature The serous coat at this point is sharply indented and there are numerous adhesions A portion of the omentum *is attached and this appears* fine and nodular and is infiltrated with gray tissue A small, hard lymph node 1½ cm in length is also found On opening the specimen, the mucosa contains two large, deep *seated, smooth walled ulcerations* measuring 2 5 cm and 1 5 cm respectively These ulcers are situated in a thick bed of tumor tissue which extends from the mucosal surface through the entire thickness of the wall The rugal pattern *of the mucosa at the margins of* the growth is *greatly altered* The tumor ex*tends to the pyloric ring, but appears to end* abruptly at that point The specimen includes the first centimeter of duodenal mucosa '

The microscopic examination revealed *mucinous carcinoma of the stomach,* with secondary carcinoma in the omentum and the regional lymph node

Roentgenographic examination (Fig 147) revealed a rather *irregularly outlined* niche at *the lesser curvature pars pylorica* The lesser curvature to either side appeared rigid, particularly the area distal to the niche giving a definite impression of *infiltration It was on* the basis of these findings that the preoperative diagnosis was made of an ulcerating malignant lesion of the pars pylorica

(1) the irregular configuration of the ulcerated area represented by the niche (2) the fact that the ulcer was within the confines of the stomach and did not project outwardly beyond the contour of the lesser curvature itself (3) the wide zone of elevation around the ulcer representing the irregularly thickened infiltrated wall of the new growth within which ulceration had developed

The importance of mucosal studies as an aid in determining the malignant nature of an ulcerating gastric lesion is further illustrated by the case that follows

A W male aged 55 Following the repair of a stab wound in the left upper quadrant 2½ years previously which had nicked the gastric serosa the patient complained of a soreness in the lower abdomen which became a gnawing ache progressing in severity He had lost 25 pounds There was occasional vomiting but no blood Physical examination of the abdomen was essentially negative Gastroscopy revealed a necrotizing ulceration of the pylorus with regional infiltration suggestive of carcinoma

Operation revealed a globular multiple tumor about 1½ inches in diameter on the lesser curvature of the stomach involving the serosa There were several glairy mucoid nodules in the center of the mass The gastrohepatic ligament was filled with round hard shotty nodes and the dome of the left lobe of the liver and the dome of the right lobe were studded with metastatic deposits of varying sizes

The biopsy of a nodule removed from the liver revealed metastatic adenocarcinoma

Roentgen examination with the stomach filled with barium (Fig 151A) showed a small area of rigidity of the lesser curvature at the junction of the pars pylorica and the pars media without definite evidence of ulceration The mucosal study (Fig 151B) showed an irregularly outlined niche at the lesser curvature projecting within the lumen of the stomach The lesser curvature both proximal and distal to the niche appeared rigid and there was a rather wide translucent zone extending across the lumen of the stomach from the lesser towards the greater curvature There were several additional rounded translucent areas at the base of the niche There was also a wide defect of the greater curvature in the pars pylorica The mucosal appearance therefore indicated a large ulcerating lesion extending into the lumen of the stomach and surrounded by abnormally prominent irregularly outlined gastric folds The evidence weighed heavily in favor of an ulcerating lesion malignant in character



Fig 153 (A *Left*) Malignant gastric ulcer (B *Center*) Same patient as is shown in (A) Seven weeks later Note the small cone shaped niche (C *Right*) Same patient as is shown in (A) and (B) Four weeks after (B) Note the marked increase in the size of the niche

The importance of mucosal studies is further illustrated by the next case

S K male aged 49 During the preceding 6 months the patient had complained of pain in the epigastrium occurring shortly after meals He was not relieved on an ulcer regimen During the 4 months before his admission to the hospital he had had tarry stools and occasional vomiting The pain finally became constant radiating to both sides and substernally He had lost 7 pounds Physical examination of the abdomen revealed an ill defined mass in the epigastrium Gastric analysis revealed essentially normal acid values

within a malignant lesion The diagnosis receives further support when the wall surrounding the niche is wide rigid and of irregular outline These features are illustrated by the following case

C M, male, aged 66 During the preceding 20 years, the patient had complained of gnawing epigastric pain occurring from 2 to 3 hours after meals and occasionally relieved by food He remained well for a number of years, the last recurrence being 1 year before hospitalization During this time the pain again occurred from 2 to 3 hours after meals and was frequently relieved by food or soda The pain often occurred at night On two occasions he noticed tarry stools lasting for 2 or 3 days He became progressively weaker and had lost 20 pounds in the preceding year

Physical examination revealed an extremely pale, elderly male Examination of the abdomen disclosed an enlarged liver and the scar of a former herniotomy There was no palpable mass

Operation revealed a large indurated crater on the lesser curvature of the stomach in its distal third measuring about 5 cm in diameter Its surface was covered by smooth reddened mucosa, which was not friable There were some enlarged nodes along the greater curvature in the gastrocolic omentum The liver was somewhat enlarged but smooth throughout A gastric resection was done

The report of the macroscopic examination was as follows "On opening the specimen a large, deep ulcer is seen on the lesser curvature in the prepyloric region Its base is smooth dense tissue On section the tissue around the ulcer is denser and whiter and about 0 5 cm thick, extending almost through the wall of the stomach The pylorus is small stenotic and friable No nodes are found"

The microscopic examination was reported on as follows "Sections show that the base of the ulcer and the underlying tissue through all layers of the stomach to the serosa are made up of a malignant tumor which consists of interlacing cords of polyhedral epithelial cells which have vesicular nuclei and large conspicuous nucleoli The edge of the ulcer shows abrupt transition into normal epithelium

The diagnosis was undifferentiated carcinoma of the stomach

The roentgen examination revealed the following When the stomach was distended with barium, there was no definite evidence of any ulcerative lesion (Fig 150A) The lesser curvature, pars pylorica, appeared rigid with a slight elevation at about its mid portion In the mucosal relief study (Fig 150B) there was an irregularly outlined, ovoid shaped niche separated from the rest of the stomach by a wide irregular translucent zone These findings were interpreted as representing ulceration within a new growth because of the following features



Fig 152 (A *Left*) Malignant gastric ulcer (B, *Right*) Same patient as is shown in (A) *Note the importance of mucosal study in demonstrating the ulcerating lesion* and the abnormality of the gastric folds

surface of the stomach The tumor infiltrates the entire wall and its surface is ulcerated The fundus is uninvolved'

The microscopic report was Sections through the stomach reveal an extensive in filtrating tumor mass This is widely ulcerated and has penetrated through from the mucosa to the serosa and the omental fat but in all cases it is capped by the latter The tumor mass is in many places contiguous with the epithelium of the mucosa The tumor is com posed of cells of varying sizes containing one or more eccentrically placed vesicular nuclei containing one or more nucleoli These cells are arranged in a tubular shape for the most part They vary in staining properties Their growth is wild and irregular The tumor is heavily infiltrated with acute inflammatory cells The blood vessels show intimal prolif eration and are congested and occasional hemorrhage into the tissue occurs The fundus of the stomach is uninvolved Along the edge of the tumor the tissue is infiltrated with chronic inflammatory cells The tumor in no portion reaches the plane of excision Diagnosis Adenocarcinoma of stomach ul cerated

Roentgen examination showed the gradual increase in the degree of ulceration of the lesion At first (Fig 153A) there was a slight elevation of the lesser curvature Seven weeks later examination showed a small cone shaped niche in this region (Fig 153B) The distal lesser curvature appeared rigid About 4 weeks later examination (Fig 153C) showed a marked increase in the size of the niche which was now rounded in contour The distal lesser curvature again appeared rigid In a period of 11 weeks there was therefore a marked increase in the size of the niche In addition the distal lesser curvature appeared rigid in the last two observations Failure to operate sooner was due entirely to the patient s procrastination operation hav ing been recommended to him because of the increasing severity of the clinical manifesta tions and the exaggeration in size of the ulcerating lesion When examined at opera tion this proved to be an ulcerating adeno carcinoma

Location of a niche on the greater curva ture of the stomach is strong presumptive evidence of the malignant nature of the lesion Even in this location detailed study of the lesion may yield further evidence to substantiate the diagnosis This is illus trated in the following case

J D male aged 62 During the preceding 3 months the patient had complained of attacks of sharp right upper-quadrant pain radiating substernally and toward the pre cordium and to the back The pain lasted for about 1 hour and was not related to meals He had lost 30 pounds Physical examination of the abdomen disclosed an irregular tender mass in the epigastrium

Operation revealed a large ulcerating lesion of the greater curvature in the prepyloric re gion A subtotal gastrectomy was done



Fig 155 Gas-capped niche of a malig nant ulcer of the stomach (verified at autopsy)

The pathological diagnosis was carcinoma of the stomach

Roentgen examination (Fig 154A) re vealed a flat niche of the greater curvature in the pyloric portion of the stomach The wall surrounding the niche was irregular in outline (Fig 154B) Apart from the fact that this lesion was on the greater curvature which in itself would favor a diagnosis of malignancy the roentgen characteristics were such that even if these findings were present on the lesser curvature the diagnosis of malignant ulcer would have been justified As an in cidental finding there was a diverticulum at the inner border of the second portion of the duodenum.

Operation revealed a large tumor mass infiltrating the lesser curvature and the pyloric portion of the stomach. There were many pyloric nodes involved with tumor tissue. The liver was studded with small hard, whitish tumor masses, ranging from 3/4 mm to 1 cm *in diameter. There was about 500 cc of thin,* yellowish fluid in the peritoneal cavity. A biopsy of the liver was taken. The pathologic diagnosis was papillary adenocarcinoma, *metastatic in the liver*

Roentgen examination (Fig 152A) with the stomach distended with barium showed a small niche of the lesser curvature, pars media. From this appearance alone it would have been difficult to state with assurance that the lesion was malignant. With a thin layer of barium, however, (Fig 152B) the destruction of the mucosa was well shown. In addition, there was a dense, fairly well circumscribed intragastric collection of barium hugging the lesser curvature pars media apparently due to an ulcerating lesion. The fact that this region did not *project out*wardly from the lesser curvature was evidence indicating the malignant nature of the ulcerating lesion, in addition to the destruction of the mucosal folds. The nichelike projection seen in Figure 152A was not noted in Figure 152B, apparently because the barium had fallen away from this region. The preoperative diagnosis was diffuse carcinoma of the stomach

Rapid increase in the size of a niche under medical observation may be suggestive of an expanding ulcerating lesion with in a new growth. This is illustrated by the case that follows

J P, male, aged 43. The patient gave a 10-year history of episodes of indigestion. During the preceding year, however, he had developed severe epigastric pain, occurring from 4 to 5 hours after meals, radiating to the back and relieved by food and alkalies. There was no nausea, vomiting or bleeding. *The patient had gained 15 pounds. Physical* examination was essentially negative except for tenderness in the epigastrium

A subtotal gastrectomy was done at which time the surgeon reported that there was an ulcerating lesion on the lesser curvature of the stomach. He suspected that he might be dealing with an early carcinoma. No obvious metastases were present

The macroscopic report was "Specimen received in Bouin's solution consists of a portion of stomach which measures 10 x 7 x 2.5 cm. On the lesser curvature near the *pylorus is a hard irregular, nodular mass* about the size of a half dollar. The rest of the serosa is smooth and glistening

On section, the following is revealed: there is an ulcer measuring 4 x 3 x 1.5 cm on the posterior portion of the stomach near the pylorus. This ulcer has sloping edges. Its floor is ragged and nodular. The wall of the lesser curvature and of the posterior portion *of the stomach in that area is replaced by* dense white glistening tumor tissue. The tumor mass extends into the serosa but it is covered by it and doesn't extend further. There are no nodes present on the exterior



FIG 154 (A *Left*) Malignant ulcer of the greater curvature pars pylorica (B, *Right*) Same patient as is shown in (A). The detailed characteristics of the niche are well shown. In addition, there is a diverticulum of the second portion of the duodenum

surface of the stomach The tumor infiltrates the entire wall and its surface is ulcerated The fundus is uninvolved"

The microscopic report was 'Sections through the stomach reveal an extensive infiltrating tumor mass This is widely ulcerated and has penetrated through from the mucosa to the serosa and the omental fat but in all cases it is capped by the latter The tumor mass is in many places contiguous with the epithelium of the mucosa The tumor is composed of cells of varying sizes containing one or more eccentrically placed vesicular nuclei containing one or more nucleoli These cells are arranged in a tubular shape for the most part They vary in staining properties Their growth is wild and irregular The tumor is heavily infiltrated with acute inflammatory cells The blood vessels show intimal proliferation and are congested and occasional hemorrhage into the tissue occurs The fundus of the stomach is uninvolved Along the edge of the tumor the tissue is infiltrated with chronic inflammatory cells The tumor in no portion reaches the plane of excision Diagnosis Adenocarcinoma of stomach ulcerated'

Roentgen examination showed the gradual increase in the degree of ulceration of the lesion At first (Fig 153A) there was a slight elevation of the lesser curvature Seven weeks later examination showed a small cone shaped niche in this region (Fig 153B) The distal lesser curvature appeared rigid About 4 weeks later examination (Fig 153C) showed a marked increase in the size of the niche which was now rounded in contour The distal lesser curvature again appeared rigid In a period of 11 weeks there was therefore a marked increase in the size of the niche In addition the distal lesser curvature appeared rigid in the last two observations Failure to operate sooner was due entirely to the patient's procrastination operation having been recommended to him because of the increasing severity of the clinical manifestations and the exaggeration in size of the ulcerating lesion When examined at operation, this proved to be an ulcerating adenocarcinoma.

Location of a niche on the greater curvature of the stomach is strong presumptive evidence of the malignant nature of the lesion Even in this location detailed study of the lesion may yield further evidence to substantiate the diagnosis This is illustrated in the following case

J D, male aged 62 During the preceding 3 months the patient had complained of attacks of sharp, right upper-quadrant pain radiating substernally and toward the precordium and to the back The pain lasted for about 1 hour and was not related to meals He had lost 30 pounds Physical examination of the abdomen disclosed an irregular, tender mass in the epigastrium

Operation revealed a large ulcerating lesion of the greater curvature in the prepyloric region A subtotal gastrectomy was done



FIG 155 Gas capped niche of a malignant ulcer of the stomach (verified at autopsy)

The pathological diagnosis was carcinoma of the stomach

Roentgen examination (Fig 154A) revealed a flat niche of the greater curvature in the pyloric portion of the stomach The wall surrounding the niche was irregular in outline (Fig 154B) Apart from the fact that this lesion was on the greater curvature which in itself would favor a diagnosis of malignancy the roentgen characteristics were such that even if these findings were present on the lesser curvature the diagnosis of malignant ulcer would have been justified As an incidental finding there was a diverticulum at the inner border of the second portion of the duodenum

Exceptionally, a niche apparently possessing the essential characteristics of a benign lesion surmounted by an air bubble proves on histologic study to be due to an underlying malignant process Such were the findings in the following case

M B, aged 31 years This patient had been complaining for 2 years of pain occurring about 2 hours after meals, the pain was sharp and was temporarily relieved by soda and food There had been periodic remissions during which he was free from distress He had lost 48 pounds in the preceding year His stools at times were pitch black Just prior to his admission to the hospital, he had had a sudden, severe attack of epigastric pain

At the time of hospitalization, physical examination revealed generalized abdominal rigidity with exquisite tenderness throughout, most marked in the epigastrium There was obliteration of liver dullness

At operation, there was a ¼ inch perforation of an apparent gastric malignancy, which perforation was located on the anterior surface of the antrum The malignancy extended well up on the lesser curvature A piece of omentum was sutured at the site of the perforation

The autopsy report was The prepyloric region of the lesser curvature of the stomach has an ovoid ulcer about 4 cm in diameter It has a perforation of 1 5 cm in diameter This is plugged by the omentum The floor of the ulcer has an eroded vessel '

The microscopic examination reported Sections reveal a large crater in which the gastric mucosa is entirely absent with overhanging edges The floor has four ill defined zones (1) an inflammatory zone consisting of fibrin and polymorphonuclear leukocytes (2) a zone of necrotic granulation tissue (3) a zone of living granulation tissue, (4) a zone of dense scar tissue

In the scar tissue forming the floor of the ulcer there are large, hyperchromatic, anaplastic cells, arranged in masses and in single columns separated by dense stroma Diagnosis Ulcerocarcinoma of the stomach There are metastases to the nodes around the epiploic region Sections through one of these nodes show that the normal architecture is markedly distorted There are broad sheets of fibrous tissue invaded by large anaplastic cells with hyperchromatic nuclei These are arranged in masses and in single columns with some glandular formations Diagnosis Metastatic carcinoma '

Roentgen examination made a few days prior to the perforation (Fig 155) showed a large niche of the lesser curvature at the junction of the pars pylorica and the pars media It was of smooth contour It contained a fluid level line surmounted by a large air bubble The lesser curvature of the stomach both proximal and distal to the niche was of smooth outline The diagnosis was large ulcer of the stomach, benign Because of the fluid level line and the air bubble above it the lesion was considered to be of the perforating type Three days later, acute perforation occurred, which necessitated prompt surgical intervention The diagnosis of the malignant nature of the lesion was made at operation and on the basis of the findings at autopsy with evidence of malignancy, not only in the ulcer itself but of metastatic invasion of a lymph node

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21

Diverticula of the Stomach

ROENTGEN DIAGNOSIS

The earliest reference to a gastric diverticulum was that by Helmont[1] Emil Hirsch[2] in 1903 described a diverticulum of the cardia of the stomach which he had noted 2 years previously at autopsy He commented on the fact that the egg shaped sac of a depth of 4 5 cm showed no scars or defects in the mucosa and that the serosa was mooth in character Up to the time of the publication of his paper, Hirsch recorded only 14 cases of gastric diverticula in the literature, including his own case

Gastric diverticula are of comparatively rare occurrence as shown not only in our own personal experience at Bellevue Hospital, but also in the records of other large clinics[3]

There are two regions that are particularly favored in the development of gastric diverticula the cardiac area and the pyloric end of the stomach The site of predilection for their development is on the posterior wall of the stomach near the lesser curvature just below the level of the entrance of the esophagus In isolated instances they have originated from the anterior wall of the stomach near the lesser curvature pars cardia[4] and on the greater curvature of the cardiac portion of the stomach[5]

The wall of the diverticulum as a rule is made up of all the layers of the stomach In some cases, the mucosa is apt to be thin and atrophic and the muscular layer poorly developed or completely absent

The theory of the embryologic origin of diverticula at the cardia is supported by the following facts First the presence of diverticula during embryonic development Thus Schwalbe[6] described a small saccular appendage on the posterior surface of the cardia in the embryonic stomach, which he described as 'diverticulum fundi or "diverticulum ventriculi ' He believed that the developmental history accounted for the persistence of such gastric diverticula in the adult Another reason in support of the embryologic theory is the fact that such diverticula are found in the stomach of the hog and certain primates[7]

That a gastric diverticulum may be present in the newborn is shown by Sinclair[8] who reported a congenital diverticulum of the stomach removed at operation in an infant 4 months old Microscopic examination showed that it had a structure identical with that of the stomach Symptoms necessitating operation had resulted from constriction of the pedicle by the transverse meso-colon through which the diverticulum had passed Sir Arthur Keith in commenting on the case stated that he entertained no doubt regarding its origin from the stomach and its congenital nature He believed that the diverticulum originated from the dorsal border of the stomach during the second month of embryologic development

Strong evidence is present to indicate that in many cases at least diverticula at the cardia are not congenital, but acquired and are the result of increased pulsion due to mechanical factors

The reason for the presence of an area of diminished resistence at the cardiac end of the stomach is explained on the basis of the following anatomic factors At the dorsal wall of the pars cardia, there is a division of the longitudinal muscle fibers One of

When ketones are produced they are first excreted in combination with ammonia As the ammonia forming capacity of the kidney is exceeded the *fixed base* of the body is withdrawn The salt loss is attended by water loss to preserve the isotonicity of the body fluids and extreme tissue dehydration may be encountered

Clinical Manifestations —The onset of diabetes is distinctly variable In young children and following acute infectious diseases the metabolic disorder begins abruptly with an almost explosive violence In middle aged and elderly patients the disturbance is often asymptomatic for long periods of time and may be revealed only as a chance finding in the course of a routine urine examination (*diabetes decipiens*)

The symptoms of diabetes are characteristic and usually uniform They consist of *polydipsia polyuria bulimia* and *pruritus* which almost invariably is localized to the female genitals The insatiable *thirst* is often attended by a sensation of *oral burning* The increased appetite has a paradoxical association with a *progressive loss of weight and strength* Those who were previously obese show the greatest evidences of weight loss Less frequent initial complaints are somnolence constipation dimness of vision impotence, muscle pains a pyoderma or a gangrenous ulceration

Dermatoses—Skin manifestations occur with regularity in uncontrolled *diabetics* Superficial *dermatophytoses* are apt to be troublesome and often lead to serious complications due to secondary infection The skin is often dry and scaly, particularly if there is dehydration and an associated *avitaminosis* *Cutaneous lipoid deposits* such as the xanthoma diabeticorum are seen about the eyelids The palms and soles may be yellow due to the high carotene content of the plasma Pyodermas such as *furunculosis* and *carbuncles* have serious implication

Digestive Disturbances—Enlargement of the liver usually the result of *fatty infiltration* is encountered most often in young children The viscus may be huge so that the abdomen is protuberant a finding of evil prognosis since it signifies a severe diabetes in which insulin reactions and episodes of acidosis are commonly encountered

The intercurrent development of acute hepatitis cirrhosis of the liver or hemochromatosis increases the severity of the diabetes and favors the development of an acidosis Tremendous doses of insulin are necessary to control the disease in the presence of these complications

Cholelithiasis (p 1997) occurs with unusual frequency in diabetics who often complain of post prandial epigastric distress intermittent pains in the right upper quadrant and recurrent attacks of jaundice In these situations there is the possibility that the gallstones served as the initial lesion and that secondarily the chronic cholecystitis and pancreatitis were instrumental in the genesis of the insular deficiency

Neurological Disturbances—The neurological disturbances that are associated with uncontrolled diabetes mellitus are now recognized as more likely manifestations of *vitamin B deficiency* They include *neuralgias paresthesias* and *anesthesias* Occasionally a *peripheral neuritis* is demonstrable and degenerative lesions of the cord produce a diabetic *pseudo tabes* The peripheral neuropathy of diabetes is usually bilateral and symmetrical It involves the lower extremities first and predominantly and rarely is reflected in the arms The usual complaint is a bilateral plantar hyperesthesia of sock distribution Often there is tenderness of the calf

muscles loss of the senses of vibration and position with absent ankle and knee jerks

Vascular Changes—The frequently encountered manifestations of cardiovascular disease in uncontrolled diabetics include *coronary sclerosis coronary occlusion cerebral arteriosclerosis* and *peripheral endarteritis* and *gangrene* The vascular changes are attributed to the disordered fat metabolism and the hypercholesteremia which is a factor of great importance in experimental atherosclerosis

Coronary occlusion (p 983) occurs twice as frequently in the uncontrolled diabetic as in the nondiabetic Acute coronary occlusion may be mistaken for diabetic coma A sudden hypoglycemia may produce angina pectoris and precipitate an acute cardiac decompensation *Arterial hypertension* is common in uncontrolled diabetes and contributes to the extent of the arterial change

Ocular Changes—Disturbances of the visual apparatus are serious and common features of uncontrolled diabetes Juvenile *cataracts* are frequent and an *iritis* may be a presenting finding Middle aged diabetics show a severe grade of *retinal arteriosclerosis* with deposits of hard yellowish refractile lipoid substance arranged in stellate figures in the macular regions Additionally there may be many small circular and linear hemorrhages seen in this area producing the so-called *central punctate retinitis* In severe diabetes the lipoid laden blood produces a peculiarly whitish waxy column on the retinal vessels (*lipemia retinalis*) *Optic atrophy* and *paralysis of accommodation* may be encountered and *decrease of visual acuity* is often a serious complaint

Renal Changes—Diabetics occasionally have a *massive albuminuria* resulting in *hypoproteinemia* and a *generalized edema* of the *nephrotic type* (p 2389) Pathological examination of the kidneys in this disorder shows a distinctive hyalinization of the glomerular capillaries constituting an *inter capillary glomerulosclerosis* (p 2372)

Laboratory Findings—Diabetes mellitus is essentially a laboratory disease *Glycosuria* is the most important diagnostic finding Sugar appears in the urine when the hyperglycemia exceeds the threshold value of 160 to 180 mg per cent In a mild diabetes the glycosuria is often intermittent so that a twenty four hour specimen must be examined It is a clinical axiom that glycosuria should be considered diabetic in origin until proved otherwise Nevertheless there are many substances which produce *pseudoreductions* in the urine (p 3675) and many other causes for glycosuria elsewhere discussed in greater detail (p 3673)

The degree of glycosuria parallels the severity of the disease if the dietary routine is kept at a constant In mild examples the twenty four hour urine sample contains up to 1 per cent dextrose whereas in the severer disturbances it may contain as much as 8 per cent

As the concentration of urinary sugar approaches 4 per cent *polyuria* is observed The polyuria is associated with a *high specific gravity* due to the extent of the sugar content In older diabetics with an elevated renal threshold for dextrose hyperglycemia may exist without glycosuria

The presence of *diacetic acid* and *acetone* in the urine is evidence of a ketotic acidosis and heralds impending coma *Albuminuria* occasionally is found and may be sufficiently severe to produce the diabetic nephrosis

The Blood—The diagnosis of diabetes mellitus should not be made until there have been confirmatory blood examinations Thereafter the blood figures are for the most part of academic importance

The initial examination of the blood in a suspected diabetic must include *fasting levels* and *curves* obtained after the ingestion of a standard sugar meal Usually the fasting blood sugar level is elevated beyond the normal figures of 120 to 150 mg per 100 cc and most often it exceed 200 mg per 100 cc Should the blood sugar figures be questionable a meal of 100 gm of dextrose in 300 to 400 cc of water is given The blood sugar levels are determined at 30 minute intervals for three hours Urine specimens are collected with blood sugar determination Under the conditions of the test there should not be a glycosuria and the fasting level of blood sugar is resumed within 1 or 2 hours In the diabetic the rise in the sugar content of the blood is excessive and the concentrations remain elevated with the appearance of a glycosuria

A variation of the classical *sugar tolerance test* has been introduced by Exton Rose For their procedure the fasting blood sugar is determined and 50 gm of glucose are administered orally At the end of a half hour another blood sugar level is determined and the second meal of 50 gm of dextrose is administered The third blood sugar is taken at the end of another half hour Using this technic it has been found that 95 per cent of diabetics have a blood sugar value of over 160 mg per 100 cc at the end of the first hour

See *Differential Diagnosis of Hyperglycemia* (p 733)

Most diabetics in addition to the hyperglycemia have a *lipemia* The rise includes increased amounts of neutral fat and cholesterol In severe diabetics the lipemia may be so extreme as to produce a milky appearance in the plasma The level of the *blood cholesterol* is so significant that many regard it as the best index of the severity of the disease Values over 400 mg per 100 cc are seen in juvenile diabetics and those whose metabolic disorder is complicated by arteriosclerosis gangrene xanthomatosis and cataracts The lipemia is apt to be associated with an elevation of the carotene content of the blood The pigments impart a yellow tinge to the skin most apparent on the palms At times the color may be sufficiently marked to suggest a jaundice The absence of a scleral discoloration in patients with carotenemia suggests the clinical differentiation The carotenemia probably represents a reduction in the capacity of the liver to convert this substance into *vitamin A* and may account also for the *avitaminosis* that is observed clinically

See *Differential Diagnosis of Hypercholesterinemia* (p 736)

Complications —It is the modern opinion that the only true complication of diabetes mellitus is the state of acidosis According to older views there was a direct relationship between diabetes and arteriosclerosis pyoderms cholelithiasis ophthalmologic disorders and the neuropathies There seemed an increased incidence of pulmonary tuberculosis The coincidental presence of diabetes and these alleged complications in many instances is the chance association of frequently encountered clinical manifestations In the remainder dietary deficiencies and the states of malnutrition engendered by a poorly controlled disturbance of sugar metabolism invited the secondary affliction

The importance of stressing these points pertains to prognosis The well controlled diabetic need dread the development of these additional burdens no more than the nondiabetic

Diabetic Acidosis—Diabetic acidosis is still *a common cause for death* despite the efficiency of insulin Most often the condition arises from diet ary indiscretions omission of the insulin dose *the presence of an inter current infection a surgical procedure endocrine imbalance* or diseases of the liver

See *Differential Diagnosis of Acidosis* (p 721)

Acidosis may supervene rapidly in any diabetic and lead to *immediate coma* It may be the first indication of an unrecognized diabetes mellitus *The onset is usually gradual and is preceded by several days* of dyspepsia irritability muscle pains and restlessness The patient gradually becomes disinterested drowsy and somewhat irrational The respirations are long deep and rapid (*Kussmaul breathing*) but there is no subjective feeling of air hunger as in dyspnea The breath has an *acetone odor* which may be *instantly apparent on entering* the sick room *The pulse is* rapid the skin dry and the pupils dilated The drowsiness leads to stupor from which the patient can be aroused but finally coma supervenes

The patient in diabetic coma is usually flushed hot and dehydrated with tachycardia and a low blood pressure The eyeballs are soft and dent easily on pressure The *urine contains* large amounts *of sugar and acetone* During treatment glycosuria may disappear before the acetonuria Less often ketones disappear or fail to appear in the urine even though still present in the blood as a result of a renal block The *blood sugar* usually exceeds 400 mg per cent but may be only moderately elevated The *car bon dioxide capacity of the blood may be as low as 10 per cent due to* reduction in the reserve alkali content of the body fluids The blood *non protein nitrogen* rises especially when the blood pre sure is low and dehydration is severe The *hemoglobin* and *cell volumes* are high due to hemoconcentration There is an appreciable *leukocytosis* u ually in the vicinity *of 25 000 white blood cells with a polynucleosis and a shift to the* left A leukemoid reaction resembling myeloid leukemia may be seen

The syndrome of diabetic acidosis may resemble other acute disturb ances The patient with nausea vomiting abdominal pain elevated tem perature and leukocytosis may appear to have an acute intra abdominal surgical emergency *and differentiation from acute appendicitis is often* difficult Diabetic acidosis may *resemble* an *acute coronary thrombosis* when patients have ill defined upper abdominal *pain* nausea vomiting hypotension fever and leukocytosis

Diagnosis—Diabetes mellitus is a laboratory diagnosis The practitioner is required to note the glycosuria identify the sugar *on at least one exam* ination and demonstrate a hyperglycemia

Treatment

The satisfactory management of the diabetic patient by his practitioner requires that the metabolic derangement be controlled by measures that are simple of execution and *within* the range of the potentialities of the cooperative person of normal intelligence

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2 Neither the degree of glycosuria nor the value for the blood sugar furnishes adequate criteria for the regulation of diabetes
3 The patient who oxidizes and stores the quantity of carbohydrate essential for his particular needs approaches the normal state whether or not he has sugar in his urine
4 The administration of sufficient protamine insulin to insure the utilization of the optimum of carbohydrate restores and maintains a metabolic state that approaches the normal
5 Patients treated with protamine insulin according to these tenets are rendered socially and economically useful despite the constant glycosuria
6 The glycosuric patients do not develop more frequent nor more severe infections When surgical measures are required their wounds heal as readily as those of patients whose urines were sugar free
7 The protamine-regulated glycosuric patients report extreme well being and maintenance of an optimum weight They are not in the constant jeopardy of a hypoglycemic reaction from an overdose of insulin as are other diabetics whose urines are kept free from sugar
8 The persistent glycosuria does not produce polyuria frequency of urination and polydipsia, provided that weight is maintained and ketosis and diabetic symptoms are controlled
9 The protamine glycosuric patients do not have a tendency to develop ketosis so long as there is maintenance of weight and freedom from diabetic symptoms
10 The continuous and constant glycosuria is compatible with nitrogen equilibrium
11 Vascular sclerosis apparently is not dependent upon hyperglycemia and does not occur more readily or more extensively in the controlled glycosuric patient

Laboratory Controls for the Management of Diabetes —The laboratory controls for the management of diabetes in private practice must be simplified to the point where they can be performed with facility by the physician and his patient These essentials are noted in Table 84

The Basic Diet —In the section on Dietotherapy a basic American diet for the person of average weight and height has been outlined (p 608) At rest such a subject weighing in the neighborhood of 150 pounds without *performing arduous labor or exercise requires approximately 1500 calories* derived from protein carbohydrate and fat The meals that are detailed provide for *75 gm of protein* (300 calories) *200 gm of carbohydrate* (800 calories) and *40 gm of fat* (360 calories) The foods yield adequate amounts of the *minerals* and *vitamins* but there is no harm in supplementing the diabetic dietary by a *multivitamin preparation* or a *yeast concentrate* to assure especially high values for the B complex (p 62?)

Modifications of the basic diet are dependent upon individual requirements Patients in the low income group may be compelled to *reduce the protein quantity* to 50 gm which is still adequate Maintenance of weight may require an *increased caloric intake* which is best provided by an initial *increase of the carbohydrate to 300 gm Later the fat content is augmented* provided that the ratio of grams of carbohydrate to grams of fat never exceeds 2 to 1 The use of a *carbohydrate fat ratio* of 6 to 1 is recommended for patients who are obese the juvenile diabetics those with cholecystitis and cholelithiasis with complicating arteriosclerotic vascular disease and those with recurrent acidosis

Special Foods —The use of special diabetic foods is discouraged There is no reason why the diabetic cannot be fed with the same products as the rest of the family

Sweetening Agents —The use of sweetening agents to taste is permis

Ideal Control—Unfortunately those who specialize in diabetes have made production numbers of the problems of therapy It is their contention that it takes three months to teach the patient to eat quantitatively During these three months there are required the coordinated efforts of physician, nurse dietitian technicians and members of the family The suggested armamentarium includes (*1*) Facilities for performing qualitative tests for the presence in the urine of dextrose and acetone or diacetic acid, (*2*) a reliable method for the quantitative determination of urinary dextrose (*3*) facilities for measuring carbon dioxide combining power and the concentration of dextrose in the blood (*4*) methods for determining the lipoids of the blood, (*5*) examination for total nitrogen and special fractions of nitrogen of the urine (*6*) examination for the non protein nitrogen of the blood for carotene and the various electrolytes (*7*) insulin of unmodified and protamine zinc varieties (*8*) hypodermic syringes and needles, (*9*) one flask of 500 cc of sterile saline (*10*) ampoules of sterile 50 per cent dextrose (*11*) ampoules of epinephrine (1 1000), (*12*) Benedict solution test tubes and alcohol lamps, (*13*) solution of ferric chloride or crystals of sodium nitroprusside and a solution of ammonia (*14*) a stomach tube, (*15*) a primer for the patient with directions and charts for preparing diet prescriptions menus and recipes (*16*) a nomogram for normal metabolism age sex height and weight in order to calculate the diet, (*17*) scales for weighing food, (*18*) measuring cylinders for fluids (*19*) weight scales for the patient

The practitioner of experience realizes the impossibility of meeting these ideal requirements He knows that diabetes is controllable by cruder methods Few patients can afford to weigh and measure their food intakes and it is a rare kitchen that can serve diabetic trays Laboratory control must be reduced to those tests that can be performed by the patient or the housewife Insulin injections must be regulated so that they do not interfere with the pursuit of the normal activities of the normal life The diet must conform to the average American intake of food and since the disturbance is controllable but not curable the medical directions cannot be too rigid or too exacting

Practical Therapy—The suggestions for therapy which immediately follow represent an attempt to compromise between the ideal and what is practicable Except for the initial diagnostic investigation the laboratory controls are within the capacities of the office laboratory and the patient of average intelligence A *basic diet* is outlined in conformance with the eating habits of the average American citizen The necessary foods are present in satisfactory quantities and a proper balance has been established Each patient is given the opportunity of dietary control without substitution therapy If this cannot be accomplished the patient is taught to inject *insulin* and an attempt is made to control the disease by the *single daily injection* of the protamine variety

In conformity with the teachings of Tolstoi a *glycosuria* is maintained in protamine-treated patients Since this method is not universally accepted particularly by those who strive for a sugar free urine its basis theories and practices are only briefly summarized

1 The satisfactory treatment of the diabetic who requires protamine insulin depends upon the quantity of glucose utilized, provided that an optimal weight can be maintained and diabetic symptoms and urinary ketones are eliminated

2 Neither the degree of glycosuria nor the value for the blood sugar furnishes adequate criteria for the regulation of diabetes
3 The patient who oxidizes and stores the quantity of carbohydrate essential for his particular needs approaches the normal state whether or not he has sugar in his urine
4 The administration of sufficient protamine insulin to insure the utilization of the optimum of carbohydrate restores and maintains a metabolic state that approaches the normal
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Modifications of the basic diet are dependent upon individual requirements Patients in the low income group may be compelled to *reduce the protein quantity* to 50 gm which is still adequate Maintenance of weight may require an *increased caloric intake* which is best provided by an initial increase of the carbohydrate to 300 gm Later the fat content is augmented provided that the ratio of grams of carbohydrate to grams of fat never exceeds 2 to 1 The use of a *carbohydrate fat ratio* of 6 to 1 is recommended for patients who are obese the juvenile diabetics those with cholecystitis and cholelithiasis with complicating arteriosclerotic vascular disease and those with recurrent acidosis

Special Foods—The use of special diabetic foods is discouraged There is no reason why the diabetic cannot be fed with the same products as the rest of the family

Sweetening Agents—The use of sweetening agents to taste is permis

sible *Saccharin* (benzo sulfinide) in ¼ to 1 grain tablets augments the palatability of many foods and beverages

Condiments—The diabetic is encouraged to use salt pepper mustards and tomato garnishings These relieve the monotony of the diet and add considerable zest to foodstuffs

Beverages—Most of the nonalcoholic beverages contain considerable amounts of sugar and they are permissible within limitations Diabetics

TABLE 84—LABORATORY CONTROLS FOR THE MANAGEMENT OF DIABETES

Tests Done by Physician

Urine

Routine

Quantitative Sugar (p 3675)

Done at first visit thereafter only a rough estimation is necessary

Acetone and Diacetic Acid (p 3680)

The acetone test suffices for most purposes The diacetic acid reaction need be done only in rare instances

Fermentation (p 3675)

Done at first visit to identify sugar as dextrose If there is a copper reduction without positive fermentation a specimen should be sent to clinical pathologist for spectroscopic examination

Spectroscopy

In doubtful cases the sugar can be identified by the use of the spectroscope

Blood

Fasting Sugar (p 3714)

Required at the diagnostic examination Thereafter the degree of hyperglycemia is of little importance If more than 200 or more mg per cent, there is little doubt about the diagnosis of diabetes mellitus

Sugar Tolerance (p 3716)

Mandatory if the fasting blood sugar is below the 200 level A normal tolerance curve suggests a renal diabetes if there is persistent glycosuria

Tests Done by Patient

Urine

Sugar

By Galatest method which requires only that a drop of urine be placed on a small quantity of the test powder

Acetone

Same as above

Weight

The body weight is of *greatest significance* Overweight patients are encouraged to reduce their caloric intake to the point where they maintain themselves in a state of slight undernutrition Lean diabetics are not encouraged to gain weight Children are to maintain a weight gain in keeping with normal standards (p 2727) Obese diabetics who do not lose weight on low calory diets may need supplementary administration of thyroid extract if the basal metabolic rate is low

who require rigid control may purchase soda pop sweetened with saccharin

Alcohol—The diabetic who is accustomed to partake of alcoholic beverages is not deprived of his small vice provided that his liquor is taken in moderation Cocktails and liqueurs however possess considerable quantities of sugar and the best drink for the diabetic is therefore a whiskey

with water or soda Not more than two normal sized drinks are permissible within a twenty four hour period

Tobacco —Unless the diabetic has evidences of peripheral vascular disease he may continue the use of tobacco in any form

Chewing Gum —Chewing gum often appeases the appetite of the diabetic and provides him with something to do when others are taking sweets

Exercise —The diabetic is urged to keep in good muscular tone flabbiness in the diabetic predisposes to infection and susceptibility more than in the nondiabetic

Insulin —Sooner or later the majority of diabetics are required to give themselves injections of insulin To this end they are furnished with special *insulin syringes* and *needles* a *syringe case* *vials of protamine zinc and soluble insulin* and ampoules of *epinephrine* (1 1000)

Special *insulin syringes* are usually of 1 cc capacity In addition to the centimeter scale many are also graduated in terms of insulin unitage The latter is apt to be confusing since the concentrations of insulin vary according to dilution and serious errors may be made *Hence the practitioner* should be sure that his patients are furnished the insulin syringe with the simpler calibrations

Diabetics who need only a single daily injection may keep the insulin syringe and its needles in a covered vessel containing alcohol Those who travel or require several daily injections are provided with a *syringe carrying case* preferably one in which the implements can be kept sterile so that boiling is unnecessary

Protamine insulin is marketed in vials with white labels and red lettering *The U 40 strength* contains 40 units to the cubic centimeter and the *U 80* contains 80 units to the cubic centimeter

Soluble insulin is marketed according to unitage in yellow red and green labeled vials *The yellow contains 20 units the red 40 units and the green 80 units to the cubic centimeter* Globin insulin is available in vials containing 80 units per cubic centimeter

The insulin equipment is completed by ampoules of epinephrine hydrochloride (1 1000) for immediate injection in the event of an episode of hypoglycemia

The Technic of Insulin Injection —Patients are taught the handling of insulin syringes so that they become adept in the self injection of the hormone They are instructed in the technic of sterilizing assembling and loading the syringes Injections are best made in different sites on the anterior and median aspect of the mid thighs after sterilization of the skin area by vigorous rubbing with alcohol Following injection the equipment is returned to the sterile container to be made ready for subsequent injections

The most important technical considerations in the insulin injection is that the needle requires introduction at right angles to the skin We prefer to spread rather than pinch the skin and render it taut before introducing the needle

The Treatment of Uncomplicated Diabetes on the Basic Diet —The diabetic who has a glycosuria but no ketosis is placed on the basic diet for a minimal period of three days The required observations are relative to the

sible *Saccharin* (benzo sulfinide), in ¼ to 1 grain tablets augments the palatability of many foods and beverages

Condiments—The diabetic is encouraged to use salt pepper mustard, and tomato garnishings These relieve the monotony of the diet and add considerable zest to foodstuffs

Beverages—Most of the nonalcoholic beverages contain considerable amounts of sugar and they are permissible within limitations Diabetics

TABLE 84—LABORATORY CONTROLS FOR THE MANAGEMENT OF DIABETES

Tests Done by Physician

Urine

Routine

Quantitative Sugar (p 3675)
Done at first visit thereafter only a rough estimation is necessary

Acetone and Diacetic Acid (p 3680)
The acetone test suffices for most purposes The diacetic acid reaction need be done only in rare instances

Fermentation (p 3675)
Done at first visit to identify sugar as dextrose If there is a copper reduction without positive fermentation a specimen should be sent to clinical pathologist for spectroscopic examination

Spectroscopy
In doubtful cases the sugar can be identified by the use of the spectroscope

Blood

Fasting Sugar (p 3714)
Required at the diagnostic examination Thereafter the degree of hyperglycemia is of little importance If more than 200 or more mg per cent, there is little doubt about the diagnosis of diabetes mellitus

Sugar Tolerance (p 3716)
Mandatory if the fasting blood sugar is below the 200 level A normal tolerance curve suggests a renal diabetes if there is persistent glycosuria

Tests Done by Patient

Urine

Sugar
By Galatest method which requires only that a drop of urine be placed on a small quantity of the test powder

Acetone
Same as above

Weight

The body weight is of *greatest significance* Overweight patients are encouraged to reduce their caloric intake to the point where they maintain themselves in a state of slight undernutrition Lean diabetics are not encouraged to gain weight. Children are to maintain a weight gain in keeping with normal standards (p 2727) Obese diabetics who do not lose weight on low calory diets may need supplementary *administration of thyroid extract if the basal metabolic rate is low*

who require rigid control may purchase soda pop sweetened with saccharin

Alcohol—The diabetic who is accustomed to partake of alcoholic beverages is not deprived of his small vice provided that his liquor is taken in moderation Cocktails and liqueurs however possess considerable quantities of sugar and the best drink for the diabetic is therefore a whiskey

tient ambulatory However marked acetonuria and the presence of acidotic manifestations require hospitalization and intensive treatment

Diabetes Complicated by Acidosis With Few or No Symptoms and a Slight Acetone Reaction—1 To the basic diet add 3 to 6 gm of *table salt* given in tablet or capsule form several cups of *salty broth* and at least 2 to 3 additional quarts of water or an *alkaline beverage*

2 If the urine test for sugar is yellow red (2+ per cent) give an additional immediate injection of *soluble insulin U 25* and be prepared to administer a large glass of *orange juice* if manifestations of hypoglycemia become manifest

3 If the urine test for sugar is green yellow (less than 1 per cent) give an additional immediate injection of *soluble insulin U 15*

4 If the urine test for sugar is negative despite acetonuria give the *orange juice* without insulin

5 When the acetonuria has been controlled resume the basic diet and the standard protamine insulin dose but seek an explanation of the acidotic complication The possibilities include errors in diet or insulin dosage fatigue emotional or physical strain the presence of a systemic infection such as an upper respiratory infection or a tuberculosis a local infection such as a pyoderm or a dermatophytosis pregnancy or the onset of a hyperthyroidism

Diabetes Complicated by Acidosis With a Heavy Acetone Reaction and Moderate Symptoms but Without Coma—1–5 same as for diabetes complicated by acidosis with few or no symptoms and a slight acetone reaction (p 3680)

6 Immediate hospitalization

7 Start an intravenous infusion of 5 per cent glucose in saline with 1 unit of soluble insulin for each gram of the infused dextrose

Diabetic Coma—1–7 same as for diabetes complicated by acidosis with a heavy acetone reaction and moderate symptoms but without coma (see above)

8 As soon as possible substitute *Hartmann s solution* for the glucose in saline The Hartmann s solution is commercially available in ampoules which must be diluted with 25 volumes of sterile distilled water saline or saline in glucose before intravenous injection

9 If the coma is profound inject 20 to 50 cc of 50 per cent dextrose using a large syringe whose needle is plunged through the rubber tubing of the infusion set

10 Inject subcutaneously *protamine insulin U 60 to 100* and at another site *soluble insulin U 20* always being prepared at any given time to repeat the intravenous injection of the 50 per cent dextrose if a hypoglycemia develops

11 Maintain body warmth with *warm blankets* but avoid electric pads and hot water bottles which may produce burns

12 Be watchful for *acute dilatation of the stomach* and *urinary retention* On suspicion perform a *gastric lavage* and a *bladder catheterization*

weight the subjective symptoms and the presence of glycosuria or ketonuria

1 Those diabetics on the basic diet who *maintain their weight lose their diabetic symptoms* and reveal *negative tests of the urine for sugar and acetone* are regarded as satisfactorily controlled at least temporarily They are cautioned to adhere to the diet and do weekly urine analyses They report to the physician upon the reappearance of glycosuria or suggestive symptomatology

2 Diabetics on the basic diet who reveal *progressive weight loss continuation of symptoms* persistence of *glycosuria* or the development of *acetonuria* require substitution therapy with insulin To this end they are provided with the necessary equipment (p 1255) and a vial of protamine insulin U-40 On each of three successive mornings before breakfast 0.5 cc of the protamine insulin yielding 20 units of the product is injected and the basic diet is supplemented by a feeding of milk and several crackers at bedtime The patient is cautioned concerning hypoglycemic reactions and carries tablets of dextrose or an orange for immediate ingestion upon the appearance of even suspicious symptoms

3 The diabetic on the basic diet with his dose of protamine insulin of U 20 who *maintains his weight* satisfactorily *loses his diabetic symptoms* and whose *urine is free from acetone and glucose* during the three day period is instructed to decrease the insulin dosage for another three day period to U 15 then U 10 and then U 5 until a glycosuria is again observed The resumption of glycosuria is assurance of protection against hypoglycemic reactions The innocuousness of its persistence has been detailed at length elsewhere (p 1252)

4 The diabetic on the basic diet with protamine insulin U 20 who remains *symptom free with satisfactory weight* and who has *glycosuria but no acetonuria* is regarded as being in a state of satisfactory control

5 The diabetic on the basic diet with protamine insulin U 20 whose *weight is not satisfactory* or who has *persistent symptoms* or *acetonuria* is unsatisfactorily controlled and his insulin dosage is increased by U 5 every third day until the conditions of No 4 (above) are accomplished If more than 30 units are required and there is a fasting glycosuria soluble insulin is used additionally in the proportion of 1 unit of soluble to 2 units of protamine

Those patients who are satisfactorily controlled as in paragraphs 1 3 4 and 5 receive instructions and admonitions regarding the possible development of hypoglycemic episodes or acidosis They are warned that vigilance must be maintained by daily and later weekly urine analyses and the monthly submission of a specimen to the physician They are cautioned against failing to report the onset of any discomfort infection or injury no matter how trivial

The Treatment of Diabetes Complicated by Acidosis—The practitioner is justified in treating asymptomatic acidosis of a mild degree with the pa

is a good likelihood that a nonreacting preparation will be found If none can be injected without allergic responses the patient must be desensitized by the intracutaneous method as elsewhere described (p 563)

Atrophy of the subcutaneous fat tissue at the site of insulin injections has been described but is a rare complication

Insulin Resistance—A high degree of tolerance to the hypoglycemic effects of insulin occasionally arises Acidosis hepatic disease infection and certain endocrine disorders decrease sensitivity to insulin and demand a marked increase in insulin requirements Interference or destruction of the hormone is attested by the injection of 400 to 2000 units in a single day during an insulin resistant phase The experiment of Houssay in the effects of removal of the anterior pituitary gland on insulin sensitivity suggests that the disturbance may be in the nature of a hormonal antagonism

The Preparation of the Diabetic for Surgery—The diabetic who is to have an operative procedure of choice is admitted to the hospital several days before the projected operation He is given a *richer carbohydrate diet* and an abundance of *salt* and *fluids* These requirements are met by administering 200 cc of orange juice with 10 units of soluble insulin between each of the principal meals with 200 cc of water and 1 gm of table salt about one half hour before the fruit juice and again at bedtime

Supplementary *basal anesthesia* is clearly indicated particularly if it is possible to do the procedure under *local or spinal anesthesia* (p 3912) *Inhalation anesthesia* has the disadvantage that the patient cannot resume normal feedings upon his return from the operating room

If an inhalation anesthesia is used an intravenous drip is established in the operating room After the first 500 cc of 5 per cent dextrose in saline solution have run in a subcutaneous injection is made of soluble insulin U 25 On the return of the patient to his room an indwelling catheter is placed in the bladder and the urine specimens are tested for acetone and sugar at hourly intervals From this point on the management is the same as for a *diabetic acidosis* (p 1257) A yellow reduction with acetonuria calls for the immediate injection of soluble insulin U 20 to 25 a green reduction requires only 10 units If the patient is sugar free orange juice is administered to assure a sufficient amount of carbohydrate for combustion The presence of glycosuria of itself need not cause concern unless it exceeds 2 to 3 per cent Under those circumstances 1 unit of soluble insulin is administered to each 1 to 2 gm of excreted sugar As soon as possible the basic diet is resumed with the preoperative dose of protamine insulin

Diabetes and Infection—The diabetic is said to be very susceptible to infections *Boils carbuncles chronic epidermophytosis acute pneumonia acute pyelonephritis* and *pulmonary tuberculosis* are stated to be more common in diabetics than in the general population These views are opposed by statistics which reveal no significant change

Infection has a profound effect on the severity of diabetes It decreases the effectiveness of insulin and often leads rapidly to coma Any sudden change for the worse in the status of a diabetic should lead to a careful search for an infective focus Urinary tract infections and tuberculosis are often obscure and escape recognition unless sought

13 Avoid complicating the clinical picture by the injection of powerful drugs such as morphine codeine digitalis caffeine strychnine *coramine*

14 Be alert to the possibility of *water intoxication* or the *bulk reaction* (p 705) due to the intravenous infusion The appearance of these manifestations requires interruption of the intravenous medication

Diabetes Control Without Glycosuria —Those who aim to keep the urine sugar free under all conditions run the hazard of the production of *hypoglycemic shock* Additionally the patient is under the constant strain of *contriving to have clear specimens* and suffers varying degrees of anguish when the tests show a reduction If as Tolstoi states the important principle of the regulation of carbohydrate metabolism resides in the amount of sugar burned and not what spills over into the urine then this refinement *is not worth the effort that it requires An exception to this statement* exists when the diabetic has associated urinary or gynecological infection Under these circumstances bactericides may be more useful in the absence of urinary sugar

Freedom from glycosuria may be accomplished by altering the diet or the dose the type or the time relationship of the insulin injection In the event of a glycosuria the amount of food may be cut down, the dose of insulin may be increased the injection may be made earlier or later with reference to meals a combination of protamine and soluble insulin or an additional dose of protamine insulin may be tried

It is our opinion that the basic diet should be maintained and the changes made with the insulin If the protamine insulin is given early in the morning and significant glycosuria occurs after breakfast it is necessary to give a dose of soluble insulin at the same time If the significant glycosuria occurs in the evening or early in the morning it is the protamine dose that must be increased

The determination of these diurnal variations requires the separate examination of several voidings and very few patients have the time or zeal to go to that amount of trouble If a second dose of insulin has to be given many patients object to the double injection since it is generally agreed that different sites are to be employed for the protamine and *the soluble preparations*

Treatment of Insulin Shock —Hypoglycemic reactions are bound to occur in the forms of diabetic management that aim at eliminating glycosuria The patient is made aware of the early symptoms which include nervousness restlessness sweating faintness and hunger The instant these make their appearance sugar candy orange juice or some other readily available form of sugar is swallowed The patient is prepared for this emergency by having preparations in pocket or handbag If there is time a subcutaneous injection of 0 5 cc of epinephrine 1 1000 is administered

Should the patient s attempts prove unsuccessful the physician injects dextrose in 50 per cent solution intravenously and gives the epinephrine subcutaneously

Insulin Hypersensitivity and Atrophy —Anaphylactic reaction to insulin can occur as with *the parenteral administration of any other foreign protein* Under these circumstances, another brand of insulin is tried and there

is a good likelihood that a nonreacting preparation will be found If none can be injected without allergic responses the patient must be desensitized by the intracutaneous method as elsewhere described (p 563)

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With the mildest systemic or local infection the diabetic is confined to bed The basic diet and the insulin dosages are continued but soluble insulin is added when indicated as in the management of the surgical diabetic or in acidosis

The development of *tuberculosis in a diabetic* makes dietary control more difficult and calls for additional dosages of insulin If the tuberculosis is sufficiently severe to require bed rest the patient has plenty of time and leisure to make urine tests and accomplish freedom from glycosuria The same principles apply when *the tubercular patient develops diabetes* during the course of his infection Metabolic control should be easily possible and the course of the bacillary process need not necessarily suffer

Diabetes and Pregnancy—Before the use of insulin the fertility of the diabetic was low and pregnancy was a rarity There was a high incidence of *abortions* and *stillbirths* Since the use of insulin pregnancy is frequent in diabetic patients Although the maternal mortality has been reduced to a level comparable to that in nondiabetics the *fetal death rate* is still high The *fetus* of the diabetic mother is usually large and may weigh 12 to 18 pounds The infant is often *stillborn* and *macerated* at birth The large size of the fetus makes delivery difficult and increases the frequency of *birth injuries* and *cesarean sections* After birth the infant is prone to *hypoglycemic seizures* possibly as a result of hypertrophy of its islet tissue *Congenital diabetes* in the offspring of diabetic parents has been observed but is rare

The pregnant diabetic may experience a sudden increase in the severity of the disease and may rapidly lapse into diabetic coma On the other hand pregnancy has been known to ameliorate the disease The phenomena observed during pregnancy in diabetics have been ascribed to the endocrine imbalance present in these patients There is evidence that an abnormal rise in chorionic gonadotropin precedes premature delivery stillbirth neonatal death sudden toxemia with hydramnios the delivery of a macerated stillborn child and the premature delivery of a mature appearing atelectatic infant which dies within a few hours after birth These accidents may be prevented by the continuous administration of *estrogen* and *progesterone* during the last two months of the gravidity

Impregnation of the diabetic may result in increased or decreased insulin resistance If the metabolic condition becomes ameliorated it may be necessary to reduce the dose of protamine insulin in order to prevent a hypoglycemic reaction If the pregnancy increases the glycosuria the dose of protamine insulin must be augmented to prevent complications in the mother and to reduce the possibility of a compensatory hyperplasia of the fetal insular tissue

The onset of diabetes during pregnancy is not uncommon but great care must be taken not to mistake the lactosuria for diabetes In the former instance the blood sugar tests are normal and the urinary reaction reveals that the reducing substance is not dextrose The principles of therapy elsewhere outlined (p 1251) need not be altered except that it is important to maintain a sense of well being on the part of the patient absence of acidosis and normal weight gain

The Neuropathies—The diabetic neuropathies are likely manifestations

of vitamin deficiency In addition to giving large amounts of the crude vitamin B complex by mouth in a yeast concentrate *thiamine chloride* is injected intravenously in amounts of 100 mg. daily Additionally nicotinic acid is provided

Diabetes and Obesity—The obese diabetic is given a diet with exceedingly low fat perhaps in the ratio of 6 gm of carbohydrate to 1 gm of fat this reduces the caloric intake and assures weight reduction An estimation of the basal metabolic rate is done so that *thyroid extract* may be ordered as indicated

Diabetes and Disease of the Gallbladder—The diabetic with gallbladder disease is treated in the manner of the obese patient Removal of the diseased viscus may have both immediate and remote beneficial effects

Diabetes and Peripheral Vascular Disease—The control of diabetes requires that extraordinary efforts be made to prevent peripheral vascular disturbances To that end the patient is given instructions as to foot hygiene The general principles are undernoted

1 Only well fitting shoes are worn Barefoot walking is forbidden
2 Tight shoes which cause pressure points are discarded
3 Loose shoes which cause friction are discarded Metal arch supports are avoided
4 Socks with darns and holes are avoided The best sock is one made of lisle In cold weather a woolen sock may be drawn over the lisle sock
5 Circular garters are avoided Adhesive plaster must not be applied to the skin
6 The shoes are laced snugly but neither too tight nor too loose The lining of the shoe is examined so as to avoid friction due to defects or folds
7 The feet are washed frequently in warm but not hot water and scrubbed with soap and water Particular attention is given to the region of the nails and the interdigital areas After washing these same areas are thoroughly dried by patting but not rubbing and powdered At bedtime it is well to anoint the feet with oil The toenails are cut square The tendency to ingrowing or incurving is prevented by gently inserting a cotton wick until the nails grow out
8 Many large communities have expert chiropodists who do excellent work in foot hygiene and who can be relied upon to refer any but the simpler afflictions to the physician All too many however take it upon themselves to attempt the treatment of dermatophytosis and other disturbances whose care is beyond their province
9 Those who suffer from cold feet are encouraged to take contrast baths (p 3790) They should sleep with bed socks or with their feet wrapped in flannel Electric pads or hot water bottles are avoided Rubbers are worn in inclement weather

Diabetes and Hyperthyroidism—The association of diabetes and hyperthyroidism is most unfortunate since the presence of either one of the afflictions adds to the gravity of the other Since the thyroidal disturb

ance is the more easily and satisfactorily remedied the patient is hospitalized and immediately prepared and subjected to adequate surgery (p 1214) With abatement and control of the hyperthyroidism the diabetes may undergo a signal improvement and even complete disappearance

During the course of the operative procedure the management of the diabetic is as previously described (p 1251) The most unusual care and precaution must be exercised however since violent changes may occur in brief periods of time particularly if there is a superimposed thyrotoxic crisis

RENAL DIABETES

Renal diabetes (*benign glycosuria diabetes innocens*) is a benign hereditary and familial metabolic defect Its essential cause is unknown and it is described in the present section merely for convenience and by way of contrast with the condition of diabetes mellitus

Clinical Manifestations —In renal glycosuria it is assumed that the permeability of the kidneys for glucose is increased More likely the defect represents an inadequate reabsorption of glucose in the renal tubules At any rate the condition is characterized by a persistent *glycosuria without hyperglycemia changes in the sugar tolerance curve* or the *symptoms of diabetes mellitus*

See *Differential Diagnosis of Glycosuria* (p 3676)

Renal diabetes is a relatively uncommon disorder that is usually recognized in the course of a routine urine analysis Sugar is present in every specimen of urine whether voided in the fasting state or after a meal Ketosis and impairment of health are not experienced and doses of insulin have little or no effect on the glycosuria

The importance of recognizing renal diabetes rests in the prognosis and treatment The patient can be assured that the condition is benign and has no promise of leading to diabetes mellitus Treatment is not required by diet or injections of insulin The latter in point of fact may be dangerous since they are prone to produce hypoglycemic episodes

CLINICAL DISTURBANCES OF THE INSULAR TISSUE

The clinical disturbances of the pancreas as a whole have been elsewhere described (p 1937) Those lesions which involve the insular tissue predominantly are important only in so far as they produce the functional disturbances of hyperinsulinemia and diabetes mellitus They include adenomas hypertrophy and hyperplasia atrophy and malignancy

Adenomas of the Insular Tissue —The adenomas of the insular tissue are recognized only when an exploration of the pancreatic tissue is made for the relief of the acute episodes of hypoglycemia The condition may be ameliorated by a removal of the neoplasm

Hypertrophy and Hyperplasia of Insular Tissue —See *Hyperinsulinism* (p 1242)

Atrophy of Insular Tissue —Atrophy and peculiar hyaline degeneration of the insular tissue may be associated with diabetes mellitus (p 1246) Nevertheless the metabolic derangement may be present without histological changes and the allegedly specific cytologic phenomena are said to be demonstrable in those who do suffer from the disease These morphological variants in no small part are responsible for the present dis

quieting views on the pathogenesis of the disturbances of carbohydrate metabolism (p 782)

Malignancy —Malignancy of the insular tissue may give rise to rapidly progressive manifestations of hyperinsulinemia or they may be associated with a rapidly progressive diabetes mellitus (p 1246)

Surgical attempts at removal are worth the effort since the prognosis is otherwise hopeless

THE ADRENAL GLANDS

The Adrenal Medulla

The adrenal medulla constitutes the *inner layer* of the adrenal glands whose structure is more completely described in the consideration of cortical abnormalities (p 1266) The dark brown medulla is soft and pulpy It is phylogenetically an *autonomic ganglion* and as such receives preganglionic fibers from the adrenal plexus and the splanchnics

Histologically the medulla consists of irregular groups of *chromaffin* cells separated by profuse networks of vascular sinusoids The term chromaffin is suggested by the brownish coloration assumed by these cells when treated with the salts of chromic acid They are colored green by ferric chloride (*Dulpian reaction*) These color phenomena are due to the presence of epinephrine the product of the medullary cells

Epinephrine Chemistry Pharmacology and Therapeutics —See p 3877

Physiology —With the discovery of *epinephrine* in 1894 attention was focussed on the adrenal medulla almost to the exclusion of the cortex It was natural to assume that the potent pharmacological effect of epinephrine particularly in vascular tension was significant in clinical mechanisms These inferences led to the interpretation of hypertension as a hyperadrenalemia and conditions of hypotension particularly the Addisonian syndromes were regarded as insufficiencies of the medullary substance Surgeons were stimulated to perform excisions of the adrenal medulla or denervations in the attempt to control hypertension Addison s disease was treated by injections and rectal and oral administrations of epinephrine

Probably no bright and shining medical hypothesis ever suffered so rude an awakening The first suspicion of the tenuous basis of the theories of medullary function followed the demonstration that the adrenal medulla was not essential to life Next the concentration of epinephrine in the blood during rest and under physiological conditions was proved to be no greater than 1 2 000 000 000 to 1 1 000 000 000 dilutions which have no demonstrable effect on the intact normal animal

Emergency Theory —Faced with these indubitable facts Cannon postulated the theory of the *emergency functions* of the adrenal medulla It was his belief that the animal during the necessity for defense or attack poured out epinephrine thus producing an elevation of blood pressure mobilization of carbohydrates increased oxygen capacity of the blood bronchiolar dilatation a shortened coagulation time and the emotional manifestations that accompany situations of stress and strain The Cannon theory suggested to the surgeons that denervation of the adrenals might be expected to ameliorate conditions such as diabetes mellitus hyperthyroidism and hypertension

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may rise to 300 systolic to 180 diastolic The patient appears anxious apprehensive and close inspection reveals erection of the body hairs (pilo motor response) A prolonged episode is poorly tolerated and may lead to profound shock (epinephrine collapse)

Laboratory Findings—During an attack the blood sugar is elevated and there may be glycosuria By perfusion experiments it has been pos sible to detect a pressor substance probably epinephrine in the blood in increased concentration during an attack Following removal of a pheo chromocytoma large amounts of pressor substance have been recovered from the tumor tissue which gives an intense chromaffin reaction

Diagnosis—The diagnosis of pheochromocytoma is suggested by the paroxysmal evidences of hyperepinephrinemia and the finding of a pal pable mass in the flank An attack may be induced by massage of the mass change in the patient s position or a dose of insulin In likely cases the diagnosis may be established by roentgen evidence of an adrenal mass and contrast visualization of the kidneys A peri renal air insufflation is of value in visualization of the tumor by x ray

Differential Diagnosis—A pheochromocytoma must be differentiated from other conditions causing paroxysmal hypertension and autonomic nervous discharges These include *autonomic diencephalic epilepsy* usually associated with a tumor of the third ventricle *idiopathic epilepsy* (espe cially in some patients with petit mal attacks) and in the early phases of accelerated arterial hypertension It is also possible that the syndrome may result from lesions causing acute transient renal ischemia

Treatment—Removal of the offending tumor produces a prompt and dramatic cure The postoperative course is often stormy and attended by the sudden onset of severe shock as soon as the tumor pedicle is clamped To prevent this the patient is given a continuous infusion of normal saline containing sufficient epinephrine to maintain the blood pressure at a nor mal level

Neuroblastomas—The neuroblastomas of the adrenal medulla are seen in children under the age of four The tumor is often bilateral and the microscopic appearance is that of a *sarcoma*

The clinical manifestations are variable In the *Pepper type* there is a *hepatomegaly* with involvement of the mesenteric lymph nodes and a rapidly fatal course in a few weeks In the *Hutchinson variety* there are metastases in the skull and orbit with resultant rapidly increasing *prop tosis* and hemorrhagic phenomena in the eye This tumor is also highly malignant and there is no available therapeutic approach

Ganglioneuromas—The ganglioneuroma is a benign medullary tumor that occurs in children and adults It is found in the brain and abdominal sympathetics as well as in the adrenal gland

The Adrenal Cortex

The adrenal cortex is vital to the normal economy and life cannot be sustained after ablation of the cortical tissue *Adrenal cortical insufficiency* gives rise to the Addisonian syndrome and *hyperactivity* produces viril ism and manifestations of hypertension Despite its protected position the cortical tissue is the site of a variety of pathologic processes which include *hyperplasia benign and malignant neoplasms infections* such as

While there is popular acceptance of the Cannon emergency theory many who have worked in this field including the senior author have never been satisfied that the concentrations of blood epinephrine can be increased to effective levels Certainly the hypothesis is not sufficiently proven to warrant surgical interference with the adrenal medulla in any of the clinical disturbances for which the procedure has been recommended and attempted In support of this critical attitude is the failure of surgery to demonstrate any consistent and significant amelioration of any of the symptoms for which the operation has been performed

A viewpoint consistent with present knowledge is one that would attest to the *pharmacological potency* of epinephrine and admit the possibility that the substance has *functional and hormonal potentialities* Beyond this lies the twilight zone of endocrinology

Hyperadrenalemia —The problem of *functional or emergency hyperadrenalemia* under physiological conditions has been discussed in the previous paragraphs The possibility that *pheochromocytomas* may be instrumental in the production of an *intermittent paroxysmal hypertension* is discussed in the later section on neoplasms of the adrenal medulla (p 1264)

Hypoadrenalemia —Until the significance of the adrenal cortical hormone became obvious it was the general belief that hypo adrenalemia was responsible for *Addison s disease* and perhaps also for certain types of *neurasthenia* characterized by *hypotension* There is now general concurrence regarding the insignificance of the role of the adrenal medulla and the great importance of adrenal cortical hormone (p 1267) To the best of present knowledge there are no clinical implications relative to deficiency or even absence of adrenal medullary secretion

NEOPLASMS OF THE ADRENAL MEDULLA

Tumors of the adrenal medulla are rarely encountered The reported varieties include pheochromocytomas neuroblastomas and ganglioneuromas

Pheochromocytomas (Chromaffinomas) —The pheochromocytoma of the adrenal is a benign encapsulated tumor that is commonly associated with the production of paroxysmal hypertension This interesting clinical syndrome is due to the liberation of an epinephrine like substance

Pathology —Pheochromocytomas are benign tumors composed of chromaffin tissue They are usually unilateral well encapsulated and cystic The tumor may completely replace the ipsilateral adrenal but at times is found in the vicinity of a normal adrenal In some cases it is a chance finding in routine autopsies on elderly patients

Clinical Manifestations —The classical clinical features of the syndrome produced by an adrenal pheochromocytoma are evidences of repeated attacks of *intense paroxysmal vasoconstriction* These include sudden pallor intense headache nausea vomiting apprehension loss of consciousness and a marked rise in systolic and diastolic blood pressures The episodes may occur once or twice daily and may last for a few minutes or as long as several hours The attacks are precipitated by sudden changes in position (bending), exercise hunger emotion and in the absence of definite cause During an attack there are well marked evidences of hyperepinephrinemia the skin is cold and pale the pupils dilated the palpebral fissures widened and the eyeballs prominent the pulse is rapid the blood pressure

ADRENAL CORTICAL HORMONES

Preparations—The first available adrenal cortical preparations were crude extracts obtained by extracting the glands with an organic solvent miscible with water They were capable of prolonging life in adrenalectomized animals They caused a retention of sodium chloride and an increased renal excretion of potassium in adrenalectomized and in normal animals Adrenal Cortex Extract (Upjohn) has been accepted in N N R

Recent chemical investigations have demonstrated that the activity of the crude adrenal cortical extracts is due to a mixture of active principles About twenty steroid principles have been isolated from the adrenal cortex These include several *androgens* (adrenosterone) progesterone and at least four compounds which can prolong the life of the adrenalectomized animal (desoxycorticosterone corticosterone dihydrocorticosterone and 17 hydroxy 11 dehydrocorticosterone) No one of these compounds can produce all the effects of the whole gland It is not known whether the cortex prepares a number of separate hormones each with a specific function or whether these steroids are derivatives of a single parent hormone and the result of its degradation during extraction and isolation

TABLE 85—THE ADRENAL CORTICAL EXTRACTS

Product	NaCl Retaining Effect	Carbohydrate Effect
Adrenal Cortical Extract N N R	Present	Present
Corticosterone	Moderate	Moderate
11 Desoxycorticosteron	Strong	Absent
17 Hydroxycorticosterone	Absent	Strong

Desoxycorticosterone Its Chemical and Pharmacological Properties—Adrenal tissue yields small quantities of this steroid The chief source is stigmasterol a sterol found in soy beans from which it is synthetized by oxidation Desoxycorticosterone is very similar in chemical structure to progesterone from which it differs by the presence of an additional atom of oxygen Esterification with fatty acids (acetic propionic) increases the potency and prolongs the action The acetic acid ester is commonly used It is ineffective by mouth and is usually administered in oil (peanut sesame) by intramuscular injection It is insoluble in water soluble in ether alcohol and propylene glycol

Desoxycorticosterone in propylene glycol may be administered sublingually Pellets implanted subcutaneously afford adequate replacement therapy in adrenal insufficiency Desoxycorticosterone has the greatest electrolyte regulating and life maintaining potency of the crystalline compounds derived from the cortex A daily dose of 1.5 mg is sufficient to maintain an adrenalectomized dog in good condition on a salt poor diet

In animals with adrenal insufficiency and in patients with Addison's disease it causes a striking retention of sodium chloride retention of water an increase in the renal excretion of potassium an increase in plasma

tuberculosis and syphilis *hemorrhagic necrosis* and manifestations of metabolic disorders including *amyloidosis* and *hemochromatosis*

Anatomy—The adrenal glands are small triangular structures lying in close proximity to the upper poles of the kidneys Occasionally both glands are fused or there may be a complete agenesis of one gland In addition to the two main structures accessory adrenal tissue predominantly cortical may be found in the peritoneal cavity beneath the right pole of the kidney in the right lobe of the liver within the solar and renal plexuses or on the posterior abdominal wall Each gland consists of a firm reddish brown *cortex* and the *medulla*

The glands are richly supplied with blood from the adrenal renal and inferior phrenic arteries The right adrenal vein empties into the inferior vena cava and the left into the renal vein The glands are innervated from the adrenal plexus via the splanchnics

The histological structure of the cortex is complex Three distinct cellular zones are recognized The *glomerulosa* lying just beneath the capsule consists of rounded groups of cells the *fasciculata* continuous with the zona glomerulosa is the largest part of the cortex and its cells arranged in radial columns are rich in lipoid the *reticularis* is a loose irregular cellular network of well-defined rounded cells with a coarsely granular vacuolated cytoplasm The cortical cells are the source of the *adrenal cortical hormone* Secretory activity appears to be under the control of *anterior pituitary hormone*

Physiology—The adrenal cortex is essential to life Its secretion is concerned with the distribution and excretion of body water and electrolytes carbohydrate metabolism the work efficiency of skeletal muscle the maintenance of a normal level of blood pressure and renal function In addition it affects the function of the gonads the normal pigmentation of the skin and the ability to resist environmental stresses such as cold and pain

Cortical deficiency results in a loss of sodium and water in the urine a depression of the blood sodium level and hemoconcentration The decrease in extracellular fluid is associated with an increase in the intracellular fluid As sodium is lost in the urine the renal excretion of potassium decreases and the concentration of serum potassium rises There is an increased sugar tolerance sensitivity to insulin a flat glucose tolerance curve hypoglycemia and a depletion of liver glycogen

The disturbances in water electrolyte and carbohydrate functions that result from cortical deficiency lead to *adynamia* indicated by a decrease in the work efficiency of skeletal muscle and to focal and diffuse signs of dysfunction of the central nervous system

Low blood pressure faulty postural adaptation and vasomotor collapse are seen in cortical deficiency These abnormalities are not due entirely to the salt water and carbohydrate defects but may result from a local disturbance of the function of the peripheral vessels or from a central disturbance of the vasomotor apparatus The ability to withstand changes in environmental temperature painful stimuli and allergic phenomena is impaired in adrenal insufficiency as is the proper functioning of the gonads and the pigmentary cells of the skin

The administration of potent cortical extracts (see p 1267) to adrenalectomized animals produces sodium retention with an elevation of serum sodium potassium diuresis with a fall in the serum potassium concentration The potassium content of muscle falls while that of sodium increases The blood volume increases and water passes from the cells to the tissue spaces The normal level of glycogenesis dependent on the conversion of protein into carbohydrate, is restored by the cortical secretion

a decrease in urinary androgen excretion is usually apparent within three weeks

Pathology—The commonest lesion productive of adrenal virilism is a *benign hyperplasia* of the adrenal cortex The disturbance may be unilateral or bilateral causing the gland to increase to three to four times its normal size *Adenomas* are rare and vary in size from a small bean to a large palpable tumor They are encapsulated firm and have a bright yellow color *Carcinomas* are more common and are likewise yellowish growths but of varying consistency The cells of these tumors if virilizing give a positive *Ponceau fuchsin* reaction At the same time the pituitary gland often reveals small basophile adenomas with a hyalinization (*Crooke's change*) The ovaries and uterus are small and infantile There may be a persistent thymus The other endocrines are not remarkable

Clinical Manifestations.—The clinical features of the adrenogenital syndrome vary with the age of the patient at the onset and the condition of the reproductive organs The most marked changes occur in infancy and childhood The principal manifestations are hypertrichosis and changes in body contour and in the female genitalia

Hypertrichosis—The appearance of an excessive growth of hair is often the first sign of virilism The hair is similar to that of the male in texture and distribution It usually begins on the face and spreads to the legs and thighs and from the pubic region to the umbilicus Patches appear on the chest and back around the nipples over the shoulders and lumbosacral regions With the full development of hirsuties there is a tendency to baldness and temporal recession as seen in the male

Changes in Body Contour—At or shortly after puberty marked alterations occur in the normal female contour Young girls give the appearance of virile and sturdy masculinity and are usually short thick set broad shouldered and deep chested with narrow hips and marked muscular development If normal puberty has occurred the changes are less marked The skin becomes coarse and dry and bears many acne lesions The pelvic girdle is narrow and has a male configuration the breasts regress or remain small and undeveloped the voice deepens and the larynx enlarges Changes similar to these are caused by the administration of testosterone propionate (p 2401)

Genital Changes—The sex reversal is revealed by enlargement of the clitoris and a decrease in the size of the labia and vagina The clitoral hypertrophy varies from a simple enlargement to a well defined penis like structure with a groove on its ventral surface There may be a definite prepuce The uterus is small and infantile and the cervix is hard and button like The vaginal introitus is difficult to penetrate At laparotomy the ovaries are usually small fibrotic and cystic The gonadal insufficiency results in a primary or secondary *amenorrhea*

Pseudohermaphroditism—The intensity of the masculinization is determined to a great extent by the age of the patient at the onset of the adrenal lesion In the most complete forms there is a *pseudohermaphroditism* (p 2381) due to increased androgenic activity during embryonic life The patient has female generative organs and male secondary sexual characteristics The infant has the appearance of a cryptorchid male with a hypospadiac penis The latter is actually a long clitoris with a urethral orifice at its base There may be no vaginal orifice or a small one opening into the urethral meatus The ovaries and uterus are present but remain undeveloped The patient is usually raised as a male is psychologi

volume and a rise in blood pressure The defect in carbohydrate metabolism characteristic of adrenal insufficiency is not affected

In normal individuals the results are qualitatively the same When given with sufficient sodium chloride moderate as well as excessive doses produce a diabetes insipidus like condition in which the sensitivity to pitressin is reduced Excessive doses lower the serum potassium concentration and lead to "periodic paralysis" Progestational effects (development of a uterine secretory endometrium and mammary growth) are produced in the female Large doses produce adrenal atrophy The effects of overdosage of desoxycorticosterone acetate in Addison s disease are mentioned in a subsequent section (p 1277)

The oxygenation of desoxycorticosterone (in positions 11 and 17) increases its carbohydrate regulating potency, but decreases its electrolyte and life maintaining effects An excess of these steroids leads to hyperglycemia and may be responsible for certain features of the adrenal cortical syndrome (see Testosterone p 2401) The steroids with carbohydrate effects are not available in sufficient quantity for clinical use

Therapeutics —Crude cortical extracts and desoxycorticosterone acetate are the only preparations obtainable for therapeutic purposes The chief indication for the use of these substances is the treatment of Addison s disease Their use has been advocated in postoperative shock burns acute infections asthenia and related adynamic states but there is little convincing evidence that they are effective in these conditions

PHYSIOLOGICAL DISORDERS OF THE ADRENAL CORTEX

Pathological variations in the physiology of the adrenal cortex produce the clearly recognizable manifestations of *hyperfunction* which causes *adrenal virilism (adrenogenital syndrome)* and *adrenal cortical insufficiency* most commonly encountered as *Addison s disease* (p 1271)

Adrenal Virilism (Adrenogenital Syndrome)

Hyperfunction of the adrenal cortex frequently gives rise to a condition of abnormal masculinization in the female (*adrenal virilism* or the *adrenogenital syndrome*) The principal feature is the tendency to sex reversal marked by the appearance of the secondary sexual characteristics of the male and the regression of femaleness In some instances the clinical picture is difficult to distinguish from *Cushing s syndrome* (p 1159)

Etiology —Adrenal virilism is caused by the overproduction of *androgenic hormone* or hormones by the cells of the adrenal cortex The basic lesion is usually a *benign hyperplasia* and less commonly a *cortical adenoma* or *carcinoma* The presence of testicular hormone is indicated by the increased urinary excretion of androgens as tested biologically and colorimetrically The responsible hormonal agent is related to but is not identical with the naturally occurring adrenal cortical factors The syndrome probably results from the persistence or reactivation of androgenic tissue normally present in the fetal adrenal cortex between the tenth and twentieth weeks of embryonic life A tumor or hyperplasia is virilizing only if it arises from this androgenic zone which has a strong affinity for the Ponceau fuchsin stain Following adrenalectomy or removal of the tumors

a decrease in urinary androgen excretion is usually apparent within three weeks

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cally a male and excretes large amounts of androgenic material in the urine A constant finding in these patients is cortical hyperplasia or a tumor of the adrenal cortex (p 1278)

Macrogenitosomia Praecox—In the prepuberal form there is an apparent transfiguration of the female into a virile male with a complete suppression of feminine functions The child is usually somewhat obese, tomboyish overgrown and has a well advanced bone age In an extreme the condition is termed *macrogenitosomia praecox* (p 1185), and corresponds to the Pellizzi syndrome of pineal tumors (p 1184) At puberty the normal female sex changes fail to occur Instead the body hair and contour are of the male type the breasts fail to develop the voice deepens and the clitoris enlarges The condition is steadily progressive

Postpuberal Virilism—The most common variety of the adrenogenital syndrome occurs after puberty The patient who passed through normal puberty experiences a change in her menstrual cycle which becomes irregular scanty and then stops entirely Simultaneously there is a progressive hypertrichosis of the male type a tendency to gain weight and masculine variations in body form voice and genitalia The appearance of these changes is associated with a period of marked emotional distress in which there is a strong sense of shame and despair As a rule the patient shuns the company of others and heterosexual libido is diminished The condition is stationary or mildly progressive when it is due to a simple adrenal hyperplasia In some prepuberal females the adrenal cortical tumor does not masculinize but causes the onset of precocious puberty with menstruation mammary development and a strong heterosexual urge

In some adult patients with an *adrenal cortical carcinoma* the clinical picture is almost indistinguishable from that of *Cushing s syndrome* (p 1159) In addition to masculinization the findings include moderate hypertension a florid complexion acne acrocyanosis purplish striae atrophicae of the lower abdominal wall and a diabetic type of sugar tolerance Hypertrophy of the clitoris is usually present and may be an important point of differentiation from pituitary basophilism (p 1159) In these patients the hormonal imbalance differs from that of other examples of the adrenogenital syndrome in adult females, since large amounts of estrogenic hormone are found in the urine

Diagnosis—A definite diagnosis of virilism emanating from an adrenal hyperplasia or tumor requires demonstration of the offending lesion by radiography or laparotomy Intravenous pyelography is of value in the preliminary localization of the site of an upper abdominal mass Large tumors produce a downward displacement of the kidneys and distort the calices The use of *perirenal air insufflation* is often of diagnostic value This procedure involves the introduction of 500 to 750 cc of carbon dioxide into the perirenal space just inside Gerota s fascia near the upper renal pole The gas serves as a contrast medium and allows the adrenal tumor to be delineated with great clarity on the radiograph

Urinary hormonal assays (p 1147) for androgens estrogens and 17 ketosteroids are of diagnostic importance An excessive excretion of androgens suggests a simple hyperplasia or carcinoma of the adrenal cortex A marked increase in the urinary excretion of estrogens has been observed in adrenal carcinomas High urinary values for androgens and 17 keto

steroids were noted in patients with Cushing's syndrome associated with adrenal cortical tumors and low values occur in those without tumor

Treatment.—In pronounced virilism an *exploratory laparotomy* is indicated for investigation of both cortical areas In simple hyperplasia the removal of the larger gland is recommended As a result of this procedure the condition becomes arrested and a striking return of female characteristics may be anticipated Adenomas and carcinomas are removed if possible In patients with an adrenal carcinoma or adenoma the contralateral adrenal may be small atrophic or absent

The patient with adrenal virilism is a *poor operative risk* and extremely susceptible to surgical shock and postoperative infection In consequence the pre and postoperative periods are managed in the manner of an impending Addisonian crisis *Desoxycorticosterone* and *cortin* are given in large divided doses 10 gm of sodium chloride and 5 gm of sodium citrate are injected by vein on the day before operation and for several days thereafter

The associate editor who does not subscribe to this routine believes that energetic preoperative and postoperative treatments with adrenal cortical preparations sodium salts and water tend to accentuate the already existent hypercortinism and may predispose to fatal accident According to his views it would seem logical to withhold sodium salts and water preoperatively and increase the intake of potassium Adrenal cortical preparations are used only when there is unmistakable evidence of adrenal insufficiency

Adrenal Insufficiency (Addison's Disease)

The clinical syndrome of adrenal cortical insufficiency was described in 1855 by Thomas Addison as a diseased condition of the suprarenal capsule" Despite the rarity of the disease in general practice its study is worthy of careful consideration since it has been the source of important contributions to the understanding of many fundamental processes pertaining to the metabolism of foodstuffs electrolytes and water

Etiology.—It is variously estimated that 60 to 90 per cent of the patients with Addison's disease are victims of *tuberculosis* of the adrenal glands Atrophy of unknown or unexplained origin accounts for the remainder many of whom may suffer from a primary disorder of hypophyseal function with secondary changes in the adrenal cortex Occasionally there have been reports of Addison's disease due to *amyloidosis syphilis metastatic neoplasms hemochromatosis acute fulminating infections* accompanied by adrenal hemorrhages as in the *Friderichsen Waterhouse syndrome* of meningococcemia *bilateral adrenal denervation* for essential hypertension or *surgical removal of an adrenal tumor* in the presence of a contralateral atrophy or agenesis of the opposite gland Acute adrenal insufficiency also may accompany *severe burns* the *shock syndrome* and *altitude sickness* in aviators (Armstrong)

A mild adrenal insufficiency has been regarded as an etiologic factor in chronic fatigue neurasthenia the involutional psychoses and essential hypotension At the present time there is no convincing objective evidence of any true relationship and *specific therapeusis* has not been consistently effectual

Pathology—The commonest adrenal lesion in Addison's disease is *bilateral tuberculosis* of the glands The lesion is seldom if ever primary and is usually the result of a hematogenous spread of the infection from the lung Extra adrenal tuberculous involvement usually is limited and subclinical but there may be evidences of active pulmonary skeletal hepatic, splenic or lymphatic invasions

Tuberculosis of the Adrenals—The adrenals are frequently enlarged firm and nodular with a mottled grayish red color The capsule is thickened and adherent to the adjacent structures The glands may be so shriveled that they are difficult to find Sections reveal the replacement of normal tissue by areas of confluent caseous nodules separated and surrounded by thin rims of yellow cortical tissue The medulla usually is completely destroyed since it is apparently less resistant to the tuberculous process than the cortex The disease probably starts in the medulla and spreads toward the periphery of the gland Areas of calcification are common and may be extensive Nests or nodules of cortical cells suggest an attempt at compensatory hyperplasia of the remaining parenchyma Tubercle bacilli can be demonstrated in most instances

To produce clinical Addison's disease there must be more than 90 per cent destruction of both glands Unilateral tuberculosis of the adrenal or partial bilateral tuberculosis of the adrenal gland is often observed at autopsy in the absense of any evidence of adrenal insufficiency The facts argue powerfully against the concepts of "formes frustes"

Primary Atrophy of the Adrenals—Primary atrophy of the adrenal is encountered with increasing frequency The origin of the lesion remains obscure but it has been variously ascribed to post inflammatory damage or a sclerotic process analogous to hepatic cirrhosis The condition is a slow degeneration of the cortex marked by the necrosis and disappearance of the cortical cells and replacement by loose fibrous tissue Attempts at regeneration are seen in small adenoma like islands of epithelium Some instances of atrophy are the late results of vascular injuries to the glands following more severe infections or toxemic states Others represent functional failure of the adrenotropic factor of the anterior pituitary

Lesions Other Than in the Adrenals—The pathologic findings in the organs other than the adrenals are not striking in Addison's disease The heart is small and atrophic, sharing in the general emaciation The coronary vessels appear tortuous and the aorta is hypoplastic The stomach and intestinal mucosa are atrophic and frequently ulcerated Often there is a generalized hyperplasia of the lymphatic tissues with persistence of the thymus as in status thymicolymphaticus Slight changes are observed in the other endocrine glands The *thyroid* frequently is the site of degenerative parenchymal change and marked lymphocytic infiltration The ovaries and testes usually are atrophic There are no remarkable changes in the parathyroids or pancreas but the basophile cells of the anterior pituitary show signs of degeneration and may be decreased in numbers

The patient who succumbs in an acute adrenal crisis shows marked hyperemia of the internal organs submucosal hemorrhages and ulcerations of the hollow viscera These findings suggest the presence of a widespread capillary injury and are related to the peripheral circulatory failure that is present before death The kidneys are frequently congested with evidences of tubular atrophy and cytoplasmic granulation Chronic pyelonephritis and renal tuberculosis are often present.

Clinical Manifestations—The *onset* of adrenal insufficiency was described by Addison in the following words The patient in most of the cases I have seen has been observed gradually to fall off in general health he becomes languid and weak indisposed to either bodily or mental exertion the appetite is impaired or entirely lost slight pain or uneasiness is from time to time referred to the region of the stomach and there is occasional actual vomiting which in one instance was both urgent and distressing It is by no means uncommon for the patient to manifest indications of disturbed cerebral circulation

Asthenia—Physical and psychic asthenia are constant early symptoms Fatigue may become so pronounced that most patients are unable to perform even light work or rise from bed This symptom is the clinical counterpart of the decreased work capacity of muscle demonstrated in adrenalectomized rats

Digestive Disorders—*Anorexia* is usually intense the patient experiencing *nausea* at the sight of food There may be a craving for salty foods and an early aversion to fats Epigastric distress bouts of vomiting and periods of alternating diarrhea and vomitings are frequent These symptoms culminate in a progressive *loss of weight* which may amount to 40 or 50 pounds in the course of a few months Peculiar episodes of *intractable diarrhea* associated with spasm of the abdominal muscles are encountered and often simulate an acute abdominal emergency

Neurological Disorders—Nervous and cerebral symptoms constitute interesting aspects of the symptom complex The patient is *apathetic* and easily irritated At times marked *apprehension* and *restlessness* are associated with anxiety The latter symptoms are usually associated with *hypoglycemia* and may precede the onset of *convulsions* or *coma*

Vertigo and *syncope* may dominate the clinical picture These are particularly marked on changing from the horizontal to the erect posture and are associated with a postural fall in systolic and diastolic blood pressures

Gonadal Symptoms—There is a loss of libido in both sexes In the female *amenorrhea* is usually present while the male is *impotent* Pregnancy rarely occurs Should the female become gravid she may experience an amelioration of her disturbances corresponding to the experimental observation that adrenalectomized animals survive longer while pregnant The favorable effects of gestation on adrenal insufficiency are apparently related to the production by the ovaries and placenta of progesterone and other steroids with cortical activity In children the onset of puberty is sometimes associated with a definite lessening of the severity of the disease and this in all probability is related to increase in gonadal activity

Physical Examination —The patient appears wan listless and chronically ill A striking feature is the *pigmentation of the skin* described by Addison as a dingy or smoky appearance or various tints or shades of deep amber or chestnut brown The intensity of the pigmentation varies in different subjects from a light tan to a dark brown that is almost negroid The patient often interprets the change as a suntan and the symptom may be disregarded for a long time The discoloration has a characteristic distribution being most intense in the areas of *normal cutaneous pigmentation* such as the nipples genitalia *pressure points* on elbows knees knuckles and belt line and the *exposed areas* of the face dorsum of hands neck and chest The palms and soles usually escape except for the creases and folds Pigmented moles and black freckles are often superimposed upon the general background of pigmentation

The general increase in pigment deposition affects the mucous membranes of the mouth conjunctiva vagina and anus About the oral cavity it appears as brown or black spots on the lips tongue gums and buccal mucosa The occurrence of pigment in these areas serves to distinguish Addisonian pigmentation from deposits seen in pregnancy carcinomatosis and in peoples of Negroid extraction Pigmentation of the skin and mucous membranes is present in almost every patient but has been absent in examples of adrenal insufficiency confirmed at autopsy In longstanding disease there may be extensive areas of cutaneous depigmentation (vitiligo leukoderma)

The relation of pigment deposition to adrenal insufficiency remains ob

Pathology —The commonest adrenal lesion in Addison s disease is *bilateral tuberculosis* of the glands The lesion is seldom if ever primary and is usually the result of a hemat ogenous spread of the infection from the lung Extra adrenal tuberculous involvement usually is limited and subclinical but there may be evidences of active pulmonary skeletal hepatic splenic or lymphatic invasions

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Laboratory Data —A mild degree of hypochromic anemia is usually present The degree of actual anemia is difficult to evaluate because of the attendant hemoconcentration There is a moderate degree of lymphocytosis which may be correlated with the general lymphoid hyperplasia or *status thymicolymphaticus*

Basal Metabolic Rate —The basal metabolic rate may be normal or elevated but more commonly it is low There is a definite intolerance to cold and a tendency to hypothermia

Blood Chemistry —Profound alterations occur in the volume and composition of the blood particularly during an acute crisis There are decreases in the concentrations of sodium chloride and bicarbonate and rises in potassium magnesium and urea levels The *nonprotein nitrogen* figure may be several times the normal The *blood cholesterol* may be elevated The fasting *blood sugar* is low and the oral and intravenous dextrose tolerance curves are flattened The hyperglycemic response to epinephrine is impaired or absent and the sensitivity to insulin hypoglycemia is increased The plasma volume is decreased

The changes in plasma *sodium* and *potassium* levels result from increased excretion of urinary sodium from failure of tubular reabsorption and a decreased excretion of potassium due to inability of the kidney to remove it from the blood The diminished *plasma volume* results from an excessive loss of plasma water and base and the movement of extracellular water into the intracellular compartments As a result of the latter the cells contain more water and sodium and less potassium than the normal See *Differential Diagnosis of Hyponatremia* (p 729) *Differential Diagnosis of Hyperpotassemia* (p 731)

Renal Function —The urine is usually increased in volume during the phase of salt and water diuresis In the terminal phases of adrenal insufficiency there is usually an oliguria with urine of low specific gravity the result of severe anhydremia and impaired renal circulation The ability of Addisonian patients to elaborate a concentrated urine is often moderately impaired There is marked delay in the diuretic response to a large volume of water administered by mouth or by vein and correspondingly adrenalectomized dogs appear to be extremely susceptible to *water intoxication*

Course —Unmodified Addison s disease runs a slowly progressive course with a fatal termination in two to four years Some patients have survived ten years or more from the onset of symptoms and others succumb in a few weeks or months Sudden death is frequent and may follow an acute intercurrent infection a surgical procedure exposure to cold or over exertion

Acute Crises —The course is punctuated by dramatic remissions and exacerbations In the *acute crisis* the patient is in a state of peripheral vascular collapse and presents the classical symptoms and signs of *shock* with marked oligemia lowering of plasma sodium and increase in plasma potassium Crises of this type have been attributed to *acute potassium poisoning* and are precipitated in adrenalectomized animals by the administration of large doses of potassium salts Abdominal symptoms during a crisis are frequent and may suggest an acute surgical emergency (p 1748)

Hypoglycemia —The Addisonian crisis occasionally is marked by a hy

scure Skin biopsy shows an abnormal amount of melanin in the cells of the epidermis The pigment probably is formed locally from a precursor of melanin (dioxyphenylalanine or dopa) and epinephrine Pigmentation also has been ascribed to disorder of the metabolism of vitamin C a normal constituent of the adrenal cortex and there is some evidence that it may be due to the abnormal stimulation of the pigment cells of the skin by an overabundance of a *pituitary melanophore hormone*

Cardiovascular Phenomena —The heart sounds are poor and there is a moderate *tachycardia* The heart is small and the pulse is weak rapid and



Fig 957 — Ink spot or melanin pigmentation of the buccal mucosa in a patient with Addison s disease

easily compressible *Hypotension* is usually present even in patients previously known to be hypertensive the blood pressure may be normal however and hypertension has been observed

In the presence of hypotension the ability to maintain an effective blood pressure in the erect posture is often impaired The tension may drop 30 to 40 mm of mercury on changing from a recumbent to an erect position This postural defect usually attended by postural vertigo and syncope may herald the approach of an Addisonian crisis

See *Differential Diagnosis of Hyponatremia* (p 729)

* Therapeutic Notes Courtesy of Parke Davis & Company

oral administration of sodium chloride in doses of 10 gm daily given as enteric coated pills

Desoxycorticosterone —The treatment of Addison s disease with desoxycorticosterone acetate a synthetic adrenal cortical steroid is beset with difficulties The steroid is not the complete adrenal cortical hormone and has no effect on carbohydrate metabolism Despite its use patients with adrenal insufficiency may continue to complain of weakness and asthenia and may have repeated hypoglycemic episodes with fatal termination Occasional patients in whom the state of water and electrolyte and carbohydrate metabolism is normal during therapy with desoxycorticosterone and sodium chloride may die suddenly with a sharp decrease of blood pressure and a rise in temperature but without the usual chemical changes

In addition to its limitation desoxycorticosterone acetate may produce a number of *deleterious effects* Excessive retention of sodium chloride and water produces extreme increases in the volume of extracellular fluid favoring the development of *edema* A rapid increase in blood volume imposes a sudden strain on the weakened myocardium of the Addisonian patient and favors acute cardiac decompensation *Myocardial insufficiency* is further aggravated by the *elevated blood pressure* that is associated with the use of the hormone Muscle weakness leading to *periodic paralysis* may be encountered if the serum potassium concentration is allowed to fall below 1 6 to 2 mg per cent Potassium chloride terminates these attacks

Desoxycorticosterone acetate is capable of prolonging the life of adrenalectomized animals and of rectifying the disturbances in water and electrolyte metabolism It is customary to start treatment with the daily intramuscular injection of 5 mg of desoxycorticosterone acetate in oil supplemented by 8 to 10 gm of sodium chloride by mouth A daily gain in weight of more than one pound during the first few days or more than ½ pound thereafter indicates excessive retention of sodium chloride and water and necessitates reduction of hormone and salt intake The optimal daily dose of desoxycorticosterone acetate and sodium chloride is one that maintains a normal level for the blood electrolytes and body weight without the development of edema or an excessive rise in blood pressure The hormone must be given daily The diet is rich in carbohydrate and a high intake of potassium is maintained to protect against potassium depletion

Subcutaneous Implants —Addisonian patients are also efficiently and conveniently controlled by the subcutaneous implantation of pellets of desoxycorticosterone acetate The pellets weighing about 0 125 mg are placed beneath the skin in the infrascapular region The maintenance dose of desoxycorticosterone is then determined by the daily injection of the hormone in oil while the salt intake is kept at a constant If symptoms of overdosage result the dietary intake of sodium chloride is reduced In the presence of untoward effects removal is indicated

Aqueous Extracts —Aqueous extracts of adrenal cortical substance are presently available Their desoxycorticosterone potency still remains to be demonstrated but they do have the advantage of rapid action and the patient may self administer the dose as with insulin in diabetes

Treatment of a Crisis —For the relief of acute adrenal insufficiency desoxycorticosterone acetate acts too slowly and large amounts of sodium

poglycemic seizure The episode is preceded by malaise and refusal of food and heralded by the onset of restlessness anxiety and disorientation Later the patient develops convulsions and coma with a marked depression of the blood sugar level

See *Differential Diagnosis of Hypoglycemia* (p 734)

Hyperpyrexia—In a certain number of patients the Addisonian death is characterized by a falling blood pressure a soft pulse and profound asthenia These changes are not related to the state of electrolyte and water metabolism they are apparently unrelated to the level of the blood sugar and fail to respond to adequate therapy with active hormonal substances There are noted a terminal rise in fever a sharp decrease in blood pressure and absence of the usual chemical changes suggesting a profound disturbance of the vasomotor apparatus

Diagnosis—The diagnosis of typical Addison's disease is easy to establish Suggestive or atypical early examples require laboratory confirmation

Salt Deprivation Test—The existence of adrenal insufficiency is confirmed by one of a variety of metabolic studies The salt deprivation test involves placing the patient on a diet containing less than 1 gm of sodium chloride per day for three days and measuring the blood sodium level on the morning of the fourth day A positive test is indicated by a fall in the serum sodium concentration from a normal level of 140 m eq per liter to 135 m eq or less This procedure is not devoid of danger since it may precipitate a fatal Addisonian crisis

Culter Power Wilder Test—For the conduct of the Culter Power Wilder test the patient is given a daily diet containing 0 95 gm of chloride 0 59 gm of sodium and 4 1 gm of potassium Water is given freely during the first day and the patient receives potassium citrate (33 mg per kg of body weight) in the afternoon On the second day the fluid intake is reduced to 40 cc per kg and the dose of potassium citrate (33 mg per kg) is repeated in the morning On the morning of the third day 20 cc of fluid per kg are given before 11 00 A M Urine is collected, for chemical study from 8 00 A M to 12 noon of the third day

In normal subjects the concentration of chloride in the four hour urine specimen varies from 15 to 150 mg per cent with a mean of 55 mg per cent In Addison s disease values of 225 to 360 mg per cent are commonly encountered In the presence of diabetes mellitus and in renal disease the test is of no diagnostic value The patient must be watched carefully for evidences of an impending crisis which necessitates its discontinuance

Treatment—The general care of the patient with Addison s disease is of great importance Adequate control is so difficult to attain and maintain that reference to a specialist is highly advisable Infections no matter how mild are carefully handled Excesses of physical exertion and mental strain and extremes of temperature are avoided Surgical procedures are hazardous and drugs are used with caution since untoward reactions are common A *high carbohydrate diet* protects against hypoglycemia (p 1245) and a high intake of *vitamins B and C* is indicated

Salt Therapy—The chief aim of treatment is to restore a feeling of well being and permit the patient to engage in normal activities This may be accomplished most simply, by the administration of *sodium chloride* Some patients are perfectly comfortable for fairly long periods with the

oral administration of sodium chloride in doses of 10 gm daily given as enteric coated pills

Desoxycorticosterone.—The treatment of Addison's disease with desoxycorticosterone acetate, a synthetic adrenal cortical steroid, is beset with difficulties. The steroid is not the complete adrenal cortical hormone and has no effect on carbohydrate metabolism. Despite its use patients with adrenal insufficiency may continue to complain of weakness and asthenia and may have repeated hypoglycemic episodes with fatal termination. Occasional patients in whom the state of water and electrolyte and carbohydrate metabolism is normal during therapy with desoxycorticosterone and sodium chloride may die suddenly with a sharp decrease of blood pressure and a rise in temperature but without the usual chemical changes.

In addition to its limitation, desoxycorticosterone acetate may produce a number of *deleterious effects*. Excessive retention of sodium chloride and water produces extreme increases in the volume of extracellular fluid favoring the development of *edema*. A rapid increase in blood volume imposes a sudden strain on the weakened myocardium of the Addisonian patient and favors acute cardiac decompensation. *Myocardial insufficiency* is further aggravated by the *elevated blood pressure* that is associated with the use of the hormone. Muscle weakness leading to *periodic paralysis* may be encountered if the serum potassium concentration is allowed to fall below 1.6 to 2 mg per cent. Potassium chloride terminates these attacks.

Desoxycorticosterone acetate is capable of prolonging the life of adrenalectomized animals and of rectifying the disturbances in water and electrolyte metabolism. It is customary to start treatment with the daily intramuscular injection of 5 mg of desoxycorticosterone acetate in oil supplemented by 8 to 10 gm of sodium chloride by mouth. A daily gain in weight of more than one pound during the first few days or more than ½ pound thereafter indicates excessive retention of sodium chloride and water and necessitates reduction of hormone and salt intake. The optimal daily dose of desoxycorticosterone acetate and sodium chloride is one that maintains a normal level for the blood electrolytes and body weight without the development of edema or an excessive rise in blood pressure. The hormone must be given daily. The diet is rich in carbohydrate and a high intake of potassium is maintained to protect against potassium depletion.

Subcutaneous Implants.—Addisonian patients are also efficiently and conveniently controlled by the subcutaneous implantation of pellets of desoxycorticosterone acetate. The pellets, weighing about 0.125 mg, are placed beneath the skin in the infrascapular region. The maintenance dose of desoxycorticosterone is then determined by the daily injection of the hormone in oil while the salt intake is kept at a constant. If symptoms of overdosage result, the dietary intake of sodium chloride is reduced. In the presence of untoward effects, removal is indicated.

Aqueous Extracts.—Aqueous extracts of adrenal cortical substance are presently available. Their desoxycorticosterone potency still remains to be demonstrated but they do have the advantage of rapid action and the patient may self administer the dose as with insulin in diabetes.

Treatment of a Crisis.—For the relief of acute adrenal insufficiency, desoxycorticosterone acetate acts too slowly and large amounts of sodium

chloride and dextrose are given by continuous intravenous infusion Massive doses of desoxycorticosterone acetate (25 mg) are injected intramuscularly and 25 cc of the aqueous cortical extract are introduced subcutaneously as outlined in the following table

AN OUTLINE OF THE THERAPY OF ADRENAL CRISIS

(After Thorn and Firor)

First Day

1st 12 hours

Place the patient in a warm bed

Inject 25–35 mg of desoxycorticosterone acetate in oil intramuscularly the total quantity being divided and injected in 4 different places

Inject 25 cc of adrenal cortical extract subcutaneously in 3 divided doses

Administer intravenously by slow drip 1000 cc of 1.5 per cent NaCl solution and 1000 cc of 5 to 10 per cent glucose solution to which 25 cc of aqueous cortical extract have been added

2nd 12 hours

Slow intravenous drip of 500–1000 cc of normal saline and 1000 cc of 5 to 10 per cent glucose solution

Fruit juices and ginger ale by mouth

Second Day

Inject 10–20 mg of desoxycorticosterone acetate in oil intramuscularly

Slow intravenous drip of 1000 cc of normal saline and 1000 cc of 5 to 10 per cent glucose solution

Frequent small feedings of fruit juice dextrose tablets and candy

Third Day

Inject 5–15 mg of desoxycorticosterone acetate

Administer 3–6 gm of sodium chloride by mouth as 1 gm enteric coated salt tablets

CLINICAL DISTURBANCES OF THE ADRENAL CORTEX

The clinical disturbances of the adrenal cortex become manifest through the syndromes of *adrenal virilism* or *adrenal insufficency* In either instance the nature of the pathologic disturbance remains inferential until an exploration is made of the glandular areas

Adrenal Cortical Adenoma

Adrenal cortical adenomas develop as compensatory phenomena in conditions characterized by the destruction of the normal elements They give rise to *adrenal virilism* when clinical manifestations become apparent

Adrenal Cortical Carcinoma

The carcinoma is the most common primary neoplasm of the adrenal cortex In the *adenocarcinoma* there are columns of normal looking cortical cells and in the *carcinoma simplex* the appearance is that of a highly undifferentiated tumor containing pleomorphic and giant cells with no particular form of cellular arrangement The growths are usually yellow and vary from solid structures to multiple cystic areas They tend to invade the surrounding tissue and extend into the renal and adrenal veins Distant metastases occur in the lungs liver and bones

The malignant adrenal cortical neoplasms are usually encountered before puberty or after the age of forty In children they give rise to an iso

sexual precocity They may also produce *heterosexual changes* such as the masculinization of the female or the feminization of the male (p 2481) Less often the manifestations are those of *pituitary basophilism* (Cushing s syndrome) (p 1150) or a *macrogenitosoma praecox* (p 1184)

The clinical variations are probably due to the protean hormone producing capacity of the various neoplasms *Androgenic cortical tumors* are most common and they produce virilism with suppression of ovarian function in adult females Should the same condition be found in the male it results in an isosexual or true precocious puberty

Estrogenic cortical cancers are less common Should they appear before puberty in the female they lead to isosexual or true precocious puberty In young males they cause feminization with gynecomastia loss of libido and testicular atrophy

Attempts at the removal of an adrenal carcinoma are usually ineffective and the patient eventually succumbs

Metastatic Carcinoma

On rare occasions metastases to the adrenal gland are sufficiently extensive to superimpose the picture of Addison s disease on that of generalized carcinomatosis Recognition of the condition is purely academic

Adrenal Cortical Tuberculosis

See *Addison s Disease* (p 1271)

Syphilis of the Adrenal Cortex

See *Adrenal Cortical Insufficiency* (p 1271)

Friderichsen Waterhouse Syndrome

The Friderichsen Waterhouse syndrome occurs in *meningococcemia* (p 211) The manifestations are those of an *acute adrenal insufficiency* occurring during the course of a fulminating meningococcus infection

See *Meningococcus Infection* (p 208) and *Adrenal Insufficiency* (p 1271)

Hyperplasia of the Adrenal Cortex

See *Adrenal Virilism* (p 1268)

Atrophy of the Adrenal Cortex

Atrophy of the adrenal cortex may follow a *systemic infection* such as occurs in the orchitis that accompanies mumps (p 480) It may also result from changes in the controlling *anterior pituitary gland* (p 1154) In any instance the symptoms are those of an adrenal cortical insufficiency (p 1271) whose course differs from that of tuberculosis in that there is less rapid progression and a greater hope of arrest of the fundamental abnormalities

Amyloidosis and Hematochromatosis of the Adrenal Cortex

Amyloidosis and hematochromatosis involving the adrenal cortex produce the symptoms of adrenal cortical insufficiency in addition to those of the more fundamental disturbance In amyloidosis the suggestive find

ings are the *hepatosplenomegaly* and the positive *Congo red test* (p 7) In hematochromatosis the urine contains *sugar* and the *liver* is greatly enlarged See p 1976

Bilateral Adrenal Cortical Necrosis

Bilateral adrenal cortical necrosis occurs in pregnancy and causes manifestations of Addison s disease

See *Pregnancy* (p 2675)

Adrenal Cortical Hemorrhage

Extensive hemorrhage into the adrenal cortical substance occurs during *shock* (p 928) and with severe burns These facts indicate the importance of dealing with either condition in the manner of an acute crisis of *Addison s disease* (p 1271)

SECTION X

THE NERVOUS SYSTEM INCLUDING PSYCHIATRY

The author is indebted to

DR LEO DAVIDOFF and DR SOL W GINSBURG for invaluable criticism and advice in the preparation of this section

CHAPTER 59

INTRODUCTION

MAN is distinguished from his fellow creatures by the further development of his cerebral potentialities He remembers articulates reads writes stores knowledge and reasons For these qualities as for all biological assets he pays a price If education is indeed an ornament in prosperity and a refuge in adversity it is equally true that education is the power that increases the capacity to suffer'

Whether or not it suits the practitioner s preferences he spends much of his active life dealing with functional disorders of the nervous system decidedly less often his problems involve organic neurology With due regard to the physician s needs the present section emphasizes the study of the neuroses and then deals with organic psychiatry and disturbances of the voluntary and involuntary nervous systems With deliberation the material has been written by the senior author in the belief that an ex position presented by a physician who lacks formal psychiatric training is more likely to appeal to and meet the needs of the practitioner

PSYCHIATRY

Psychiatry is the branch of the medical sciences dealing with disturb ances of thought feeling and conduct It is concerned with processes that upset the mental moral behavioristic and social aspects of an individual s personality adjustments Such disturbances may affect and modify his life work and happiness or the welfare and well being of his family and asso ciates

INCIDENCE OF PSYCHOGENIC DISTURBANCES

Each mental act is accompanied by psychic and emotional overtones of varying intensity Fear anxiety and/or apprehension accompany pain injury infection or surgical procedures no matter how trivial The ordi nary mental and emotional phenomena of obvious causation offer no im portant clinical problem The realm of psychiatry is entered when the effect is disproportionate to the cause the etiology obscure or the mani festations persistent

Psychiatric disorders of sufficient intensity to warrant medical consul tation are estimated to constitute 30 to 50 per cent of patient material observed in private practice More graphic and definitive evidence of the appalling incidence of mental disease is afforded by institutional figures These reveal that from 12 to 20 per cent of all patients admitted to gen eral hospitals present conditions and problems that are primarily psycho genic approximately one person in twenty becomes a patient in a mental hospital at some time or other during his life and one individual in ten becomes more or less incapacitated by mental disease at one time or an other whether or not he is hospitalized

PSYCHIATRY AND THE PRACTITIONER

In days gone by it was the clergyman whom the troubled sought in the hour of need In more recent times with the inclusion of applied psychology and psychiatry as integers in the practice of medicine psychogenic and emotional disturbances have become the province of the family doctor In the span of most lives the individual brings to his medical advisor problems of anxiety and concern relative to personal business and social adjustments, mental conflicts, sibling rivalry adolescent adjustment preoccupation with the care and rearing of children sexual difficulties marital and parental schisms, abnormal habits and disturbing proclivities and aberrations in behavior, feeling or thought Even the best integrated individual finds himself at some time or another, in conflict with himself his fellows or his community

The practitioner functions as a psychotherapist in most of the emotional and psychogenic manifestations that result from the ordinary tribulations experienced in the course of the average life Each form of therapy has an element of psychiatric guidance and the management of psychiatric disorders represents a major problem in private practice

The confidences brought from the patient to the practitioner afford an insight into character and thought processes They permit the compleat practitioner to acquire a more intelligent and rounded concept of clinical disturbances In exchange for the weary hours and tremendous energy expended in the consideration of the emotional problems of his patients the family doctor is given a panoramic view of clinical medicine that is in sharp contrast to that of the specialist whose shaft vision focuses on a field of less and less' breadth with 'more and more acuity

MISCONCEPTIONS REGARDING PSYCHIATRY

Among the many deterrents which handicap the practitioner in psychiatric guidance are the vast ignorance misinformation and repugnance of most of the population toward mental disease The very thought or statement that a person has a mental affliction stirs the animosity of patient and family Most individuals even in the group of the intelligent regard mental disease as something reprehensible and culpable Until one has had experience with a psychiatric disorder it is difficult to believe that these derangements are involuntary serious incapacitating or of major significance

It is unfortunate that many physicians share the opinions of the laity The patient who is neurotic is often stigmatized and berated or his illness is minimized and passed off with such expressions as he is just a nut' 'nothing but a neurotic or a hypochondriac or some similar derisive comment The patient is left with the impression that nothing is wrong or that his illness is imaginary or controllable at will Therapy frequently consists in such glib advice as snap out of it or you ought to be ashamed of yourself and other admonishments to the effect that the sufferer must not coddle himself and that in reality all he needs is a swift kick in the place where it will do the most good

Psychiatrists and mental hospitals are usually not rated on a much higher plane than the mental patient Most psychiatrists are regarded as being a bit on the balmy side The concept of the mental institution is

till that of Bedlam, with chains bars and walls with uniformed keepers who beat and abuse the inmates and with inhabitants who imagine they are Napoleon or the Queen of the May This popular but highly inaccurate picture is completed by the estimate of the hopelessness of prognosis since most lay persons and many physicians regard mental disease as incurable and therapeutic efforts as mere gestures except for custodial or penal care

Perhaps nowhere in the realm of medicine is there such a wide discrepancy between popular misconception and actual truth Often the practitioner functions in no more useful capacity than that of attempting the education of patient and community in the accomplishments of psychiatry

SOCIETY AND THE MENTAL PATIENT

Mental afflictions impinge upon society in respect to marriage divorce and child bearing

Marriage —It is apparent from the frequent familial occurrence of nervous and mental disturbances that marriage into a mental family is unwise and often terminates disastrously for the more normal marital partner It is not at all uncommon for the physician to be consulted by the spouse of a neurotic or psychotic patient since the afflicted has learned to live with his psychogenic complaint while the marital partner suffers inordinately Impotence in a husband produces serious symptoms in the more normal wife a frigid wife is the cause for many of the complaints of her integrated husband many persons have compulsions relative to sexual intercourse and these prove irritating and finally intolerable to the unfortunate husband or wife

Many marriages are made on a neurotic basis merely because the bride to-be bears a resemblance to the mother of the groom or the groom to the bride s father Such marriages begin unfavorably and are prone to lead to difficulties most particularly on a psychosexual basis

One of the questions most commonly asked the practitioner concerns marriage to a psychotic or a recovered psychotic patient There are unfortunately no available statistical data on this point The patient with *manic depressive psychosis* who desires to marry is warned of the risk of recurrence *Recovered schizophrenics* are advised not to marry and if they do they are urged against assuming parenthood In epilepsy it has been estimated that there is a 1 to 40 chance of epileptic offspring and the risk should be explained to the contracting parties

Regarding consanguinity the wisdom of the marriage of first cousins is often debated If there is no appreciable incidence of mental or nervous disease there would seem to be no medical objection

Pregnancy and Childbirth —Pregnancy and childbirth give rise to serious problems particularly in *epileptics* and *psychotics* This aspect has been recognized by many states where sterilization is *permitted in* the more serious psychoses

The practitioner is more commonly confronted with the problem of pregnancy in a *neurotic* This event is apt to be accompanied by *an ambivalence* in which the desire for maternity or paternity is opposed by anxieties and fears relative to the hazards of parturition The physician must take a firm stand against pressure brought upon him to recommend

termination of the pregnancy unless he has the fear that a psychotic episode is imminent Under these circumstances he is bound to consult the specialist psychiatrist before recommending a therapeutic abortion

SURGERY AND THE PATIENT WITH A MENTAL DISORDER

Surgical problems give rise to serious dilemmas in cases of neurotic or psychotic patients Diagnostic problems are considerably complicated particularly in the *conversion* and *transference neuroses* Under these circumstances the practitioner runs two risks which are diametrically opposed If he credits the hysterical symptom as a manifestation of a somatic disorder he may sanction or perform an unnecessary technical procedure On the other hand if he expresses too great doubt concerning the possible presence of organic disease the patient may suffer inordinately because of loss of valuable time in the execution of a necessary surgical procedure such as appendectomy or the suture of a perforated ulcer

Anesthesia gives rise to many problems in neurotics and psychotics Some demand local anesthetization in major surgery since they fear to be put asleep" Others refuse local procedures and demand inhalation anesthesia for minor procedures Exceptionally stoics refuse any anesthesia and demand that they be permitted the masochistic pleasure of enduring exquisite agony '

The *postoperative course* in the neurotic and psychotic is often a tremendous problem for physician and surgeon alike Postoperative psychoses often terminate tragically dressings are torn off catheters and tubes are pulled out and the nightmare ends with the patient leaping out of the window Oftentimes there is inordinate complaint of pain and the postoperative course is complicated by the effects of the excessive administration of drugs particularly the opiates which add obstipation and urinary retention to other difficulties All too frequently the neurotic patient will suffer from intractable vomiting paralytic ileus persistent and repeated urinary retention or a myriad of other manifestations sufficiently numerous persistent and annoying to try the patience of a saint At times it becomes necessary for the practitioner to sit down with his patient and explain that there are certain discomforts which must be endured with fortitude and courage that no further sedative medication will be ordered except for major discomfort and that unless there is more active cooperation the situation gives promise of terminating seriously if not fatally *Drug addictions* which initiate from painful illnesses are laid at the door of the physician but fundamentally they complicate a preexistent neurosis

Conscious of these surgical difficulties the practitioner looks with dread upon surgical procedures of *election* (p 3996) particularly those which the technical enthusiasts recommend for the relief of manifestations of conversion or transference These latter include appendectomy for all manner of *abdominal discomfort and correction of uterine malpositions* and gynecologic reparative procedures designed for the relief of asthenia pain in the back sexual debility and the like Paradoxically in operations of emergency the neurotic to the astonishment and delight of all concerned often comes through with a stoicism that testifies to the rela

the courage of these patients when faced with reality situations of conscious comprehension

THE INVOLUNTARY NERVOUS SYSTEM

The involuntary nervous system is the most vitally important and most neglected subdivision of the human mechanism Its significance rests in its widespread control of visceral hormonal and metabolic processes its neglect is due to the perfection of its mechanism inadequate anatomical representations in dissecting and operating rooms the rarity of demonstrable organic disease and the difficulties of approaches to therapy

Efficiency of the Involuntary Nervous System—The efficiency of the involuntary nervous system is one reason why its effects are so little appreciated The physician takes for granted its near perfection just as civilized man without due gratitude or reverence accepts the marvels of electricity radio reception and modern plumbing a switch is thrown and a bulb is expected to give out light dials are twisted and sound waves are brought in from great distances, a spigot is turned and water from a mountain side fills a bowl or bath tub Such miracles are the normal disturbances are regarded as vexatious and baffling and except for the reattachment of a loose wire or the tightening of a screw these momentous physical phenomena are quite beyond ordinary control

Many mundane observations attest to the efficiency of the involuntary nervous system It is already functioning at birth in contrast to the tardy development of the voluntary system which is responsible for the child's being virtually helpless for the first eight to twelve months of life At the other extreme when the voluntary nervous system has ceased to function the faithfulness and hardiness of the involuntary nervous system again become manifest the vegetating moribund patient afflicted with a fatal carcinomatosis may lie comatose for days or weeks before the involuntary nervous system surrenders its last spark of vitality The voluntary nervous system is put to sleep each night while the involuntary nervous system continues its ceaseless vigil There is even the possibility that medullary centers controlled by the involuntary nervous system determine the sleep walker activities of the cerebrum Small wonder then that the physician takes for granted the dependability of the involuntary nervous system in the manner of his acceptance of the law of gravity

Associations and Coordinations—The major associations and coordinations of the involuntary nervous system are not approached in their complexity and efficiency by any piece of machinery devised by man They are not alone automatic but they are self regulating to a degree that is incomprehensible Their minor functional disturbances that enter clinical consciousness in the nature of *autonomic imbalance* (p 1395) are essentially nuisance phenomena and only rarely incapacitating

An adequate exposition of the scope and interrelations of involuntary activities would require something more than the usual type of presentation A satisfactory portrayal might be attempted by utilizing the conductor's score of a great symphony In place of inscribing the notes of the scale for strings wood winds brasses and tympany the symphony of the involuntary nervous system would be scored simultaneously for the circulatory digestive endocrine respiratory reproductive urinary and meta

termination of the pregnancy unless he has the fear that a psychotic episode is imminent Under these circumstances he is bound to consult the specialist psychiatrist before recommending a therapeutic abortion

SURGERY AND THE PATIENT WITH A MENTAL DISORDER

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connect medulla with pons and cerebellum The principal longitudinal tracts are the pyramidal the posterior or median longitudinal bundle and the median fillet The superficial origins of the sixth to the twelfth cranial nerves originate or terminate in the substance of medulla and pons Reaction patterns arising from medullary involvement appear with material on brain tumors (p 1419)

The Ventricles—The ventricles of the brain contain cerebrospinal fluid Each is lined by an ependymal membrane The four cerebral ventricles are the right and left lateral ventricles situated within the hemispheres the third ventricle between the optic thalami and the fourth ventricle in the hindbrain Each lateral ventricle is connected with the third ventricle by a foramen of Monro third and fourth ventricles are joined by the aqueduct of Sylvius The fourth ventricle communicates below with the central canal of the cord and with the subarachnoid spaces through the foramina of Magendie and Luschka

Disturbances of the ventricles and their attendant reaction pictures are described with the material on hydrocephalus (p 1109) and brain tumors (p 1419)

The Spinal Cord Spinal and Cranial Nerves—The anatomic descriptions of spinal cord spinal and cranial nerves appear in the section on the peripheral neuropathies (p 1471)

The Meninges—From within outward the structures of the voluntary nervous system are invested by pia arachnoid and dura The pia is closely attached to brain and cord and is separated from the arachnoid by the subarachnoid space in which cerebrospinal fluid circulates Arachnoid and dura are eparated by the subdural space which likewise contains cerebrospinal fluid The dura finally is separated from the periosteum of the skull or vertebrae by loose areolar tissue which further functions for the insulation of the delicate neurological structures Disturbances of the meninges are discussed in the sections on meningitis (p 1462) and in the material on vascular injuries (p 1445)

The Cerebrospinal Fluid—The cerebrospinal fluid acts as a water bed for the protection of the outer surfaces of brain and cord It flows from lateral ventricles to third and fourth ventricles through the central canal of the cord and within the subarachnoid and subdural spaces

Cerebrospinal fluid may be collected by lumbar cisternal or ventricular puncture as elsewhere described (p 3736) its physical chemical and serologic characteristics are enumerated in the section dealing with laboratory diagnosis (p 3734)

bolic functions Beyond this the following important distinctions would prevail the harmony of the coordinating agencies would be assured by mechanisms of self regulation there would be no rests intermissions or intervals, provisions would be available for tightening loosening and replacing strings without interruption of the performance and the maestro would be invisible

THE VOLUNTARY NERVOUS SYSTEM

The voluntary nervous system is mainly concerned with the relationship of the individual to his external environment The spinal cord with its spinal nerves has motor and sensory functions The brain is the seat of consciousness and memory, it contains the receptive nuclei for sensory impulses which come from skin joints muscles and organs of special sense it may originate motion and control or coordinate the actions of muscles which are primarily innervated by cells in the spinal cord and the lower brain centers The brain has the faculty of correlating the knowledge which is acquired by experience and of utilizing these deductions for synthetic mental processes

The component parts of the brain are the cerebral hemispheres mid brain cerebellum pons varolii and medulla oblongata

The Cerebral Hemispheres —The cerebral hemispheres are of large size they exhibit a high degree of specialization with regard to function the reaction patterns produced by localized lesions are discussed in greater detail in the consideration of *neoplasms of the brain* (p 1423) and *vascular accidents* (p 1439) The cerebral hemispheres are connected by the corpus callosum which functions for the coordination of right and left subdivisions

Midbrain —The midbrain connects those portions of nerve tissue below tentorium cerebelli and cerebral hemispheres It is traversed by the aqueduct of Sylvius and contains structures concerned with visual oculomotor auditory, painful and caloric sensations as well as the nuclei and roots of origin of the third fourth and part of the fifth cranial nerves The reaction patterns' of midbrain injuries are discussed in the section on *cerebral neoplasms* (p 1423)

Cerebellum —The cerebellum has a central vermis and two lateral hemispheres Each hemisphere is connected with the brain stem by inferior middle and superior peduncles The cerebellum correlates sensory impulses from the internal ear muscles and other organs The reaction patterns resulting from cerebellar injury are discussed in the paragraphs on *brain tumors* (p 1423)

Pons Varolii —The pons varolii lies between midbrain and medulla it forms a bridge which connects the two cerebellar hemispheres and its posterior surface forms the upper half of the floor of the fourth ventricle It contains nuclei of origin or termination of the fifth sixth seventh and eight cranial nerves Disturbances of the pons Varolii give rise to characteristic reaction patterns described in the section on *brain tumors* (p 1423)

The Medulla Oblongata —The medulla oblongata is a direct continuation upward of the spinal cord It contains important and vital cardiac vasomotor and respiratory centers Longitudinal bundles of nerve fibers

toire of five or six words bladder and rectal control are complete and the hours of sleep have been reduced from 20 per day at six months to perhaps 15 the cup is used for drinking purposes and a spoon for the ingestion of soft foods

By the second birthday the child can run and is able to combine words into phrases its coordination is sufficient to permit it to throw a ball and make an effort at catch

At *three years* of age the child has advanced to the stage where it assists in dressing and may even put on its own shoes It recognizes the obvious portions of its anatomy such as the eyes nose and mouth and points them out in the mirror or in pictures It begins to repeat sentences and numerals and recognizes and enunciates its own name

At the age of *four* the child knows its sex recognizes common objects and may be sent on errands in and around the house At *five* it is able simultaneously to carry out several commissions draws and copies in the familiar picture books and is able to dress and undress without assistance

At *six* the child knows the time of the day recognizes pictures that are attractive and distinguishes those that are ugly appreciates the difference between the right and left hands and is able to count through the teens He describes pictures of familiar objects, names the colors and the days of the week and begins to recognize the rights of other members of the household

School Age to Adolescence.—From school age until adolescence the development and mental progress of the child are best estimated by the *scholastic standing* The school record reflects the child's intellectual capacity and behavior and his ability to cooperate and play with contemporaries of both sexes

Girls develop *secondary sex characteristics* at the age of from seven to ten and the menarche occurs on or about the age of thirteen Boys lag somewhat behind in maturation and their masculine hairiness and manly voice do not become apparent until the twelfth to sixteenth years

Without the aid of complicated and intricate tests (*q v*) the practitioner recognizes which of "his children" are developing normally in school in extrascholastic activities and in the business of adaptation to the science of living He has no difficulty in noting the backward the maladjusted misfits and unhappy youngsters Without much formality he usually has a clear picture of the etiology of the disturbance whether organic characterologic or situational—in the latter instance most likely due to parental ignorance or misfortune See *Mental Deficiency (Feeblemindedness)* (p 129)

The Normal Adult Personality and Behavior Patterns.—The concept of the normal adult is purely relative and cannot be defined with rigidity Normality varies in different cultures and at different levels of economic and social integration Mutations occur with the sex and age of the person and the position in life

It is accordingly difficult to catalogue and analyze the normal personality and behavior patterns as they are observed in clinical practice Each practitioner is well acquainted with the customs and habits in his own community and recognizes deviations therefrom Aberrations require definition for diagnostic prognostic and therapeutic purposes A child or an adult may prove to be backward or recalcitrant through no conscious desire to be difficult but as the result of mental deficiency impaired perception a defect in hearing or vision and through inattentiveness or speech impairment. The practitioner *attempts to analyze* psychic and emotional aberrations in much the same manner as disturbances in respiration or circulation

The ingredients that enter into personality behavior patterns and conduct include *mentality perception attention consciousness thought production thought progression orientation judgment memory* and *effects* Variations and disturbances may be reflected in *total personality or character alimentary or sexual appetite* and *behavior sleep visceral function motor behavior speech* or *posture* Each of these elements is defined and the disturbances catalogued in the material that follows

The "Normal" or Integrated Personality—

1 The normal individual is capable of achieving a feeling of self security and *self esteem without exaggeration in either direction*

2 He is able to work fruitfully at his given occupation and derive *satisfaction from it* He progresses with time to gain a satisfactory reward both economically and otherwise

3 He is able to maintain a pleasant and adequate relationship with his colleagues

4 He is able to give and accept love from a suitable partner and to have satisfactory sexual relationship with his mate

CHAPTER 60

PSYCHIATRY: NORMAL AND ABNORMAL MENTAL AND EMOTIONAL DEVELOPMENT

NORMAL MENTAL AND EMOTIONAL DEVELOPMENT

AN UNDERSTANDING of the variations that come into the realm of psychiatry requires a prior knowledge of the normal development and range of the intelligence and emotions. Mental aberrations and afflictions then become clearer as variants of the normal rather than processes which arise without meaning or reason.

The development of the human mind and character provides a fascinating spectacle with infinite variations on the theme of human metamorphosis. Although the definition of the normal is broad and loose, some generalities are recognized in attempts to appreciate conditions of arrested development, regression and deterioration.

At Birth and First Year.—*At birth* the child is capable of moving its hands and feet; it is frightened by sudden loud noises; it detects the odor of milk, makes its way to the source and suckles the maternal breast or the rubber nipple. It resists restraint and withdraws the part that is exposed to painful stimulus.

By the end of a month the child blinks when startled. It follows familiar objects with its eyes and in another month apparently recognizes familiar objects such as the bottle and familiar faces, particularly of its mother. *At four months* it reaches for the familiar object and apparently recognizes voices. *By the seventh month* it sits erect with slight support, vocalizes syllables and makes crowing sounds. *By the tenth month* the child sits alone and says mama or dada and perhaps learns a simple nursery trick such as shaking bye bye. *At the end of a year* the child walks with some assistance and is able to say one or two words. It begins to have an idea of obedience and should have learned sphincteric control of bladder and rectum.

Pre-school Age.—*At a year-and-a-half* the child is able to climb stairs and has a reper

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2 He is able to work fruitfully at his given occupation and derive satisfaction from it He progresses with time to gain a satisfactory reward both economically and otherwise

3 He is able to maintain a pleasant and adequate relationship with his colleagues

4 He is able to give and accept love from a suitable partner and to have satisfactory sexual relationship with his mate

5 He has intimate friends of the same sex
6 He enjoys a gratifying avocation such as a sport or hobby
7 He is capable of a certain amount of self-criticism and self-examination He sets neither too high standards for his station in life nor is he too compulsive in his own behavior
8 He is able to confront himself with his own inadequacy and transgressions without excessive anxiety or approach
9 He is ambitious but not excessively so His ambition is not such as to turn each failure into a tragedy or to compel him to make a virtue of necessity
10 He is able to take orders from superiors and give them to inferiors—in either case without hostility or resentment
11 He is able to withstand without excessive anxiety a fair amount of deprivation and loss
12 He performs his excretory functions without guilt or anxiety

ABNORMAL MENTAL AND EMOTIONAL REACTIONS

"All the world is queer save thee and me
And even thou art a little queer
Robert Owen (1771–1858)

The broad concepts of normal behavior and mental and emotional reactions add to the difficulty of defining what is abnormal The practitioner recognizes two guiding principles in his evaluation of patients with aberrations of behavior

1 The abnormal represents a *quantitative variation* from the normal
2 The borderline between normality and the abnormal is crossed when the patient suffers *interference* with life-work, companionship or the pursuit of happiness as the result of excessive or protracted mental or emotional disturbance

Mentality

The mentality of a patient reflects intellectual capacity It is independent of knowledge or education since many savants and pedants are psychopathic while illiterates may have a rich potential mental capacity See *Psychometric Tests* (p 1325)

Amentia —Amentia signifies an innate mental deficiency or a confusional psychotic state bordering on stupor and characterized by clouding of the sensorium difficulty in grasp and retention and disorientation Because of the confusion in the varying uses of this term it is best avoided

Dementia —Dementia is a blunting or restriction of mental activities and capacities There is usually growing incapacity for appreciation and learning memory is defective and confusion may exist It is increasingly difficult for ideas to penetrate and judgments to be formed especially in moral and aesthetic spheres

Feeblemindedness —Mental deficiency is usually considered present in any individual whose intelligence quotient is below 70 The grades of feeblemindedness range from idiocy through imbecility to moronity See p 133?

Perception

Perception is that mental function by virtue of which impressions made upon the sensory organs are received and identified understood or interpreted in consciousness Normally a nervous impulse produces visual auditory or other sensory images which the individual recognizes and interprets in the light of experience

Hallucination —Hallucination is an imaginary sensation usually referred to the senses of hearing sight or smell Auditory hallucinations are most commonly encountered The hallucination is a normal function in dream life

Although imaginary hallucinated material is a real part of the patient s mental life and expresses unconscious material which has broken into consciousness The material of the hallucinatory experience expresses basic emotional needs such as self criticism and punishment substitutions for love or deprivation of love or enhancement of self esteem Hallucinations are found in many serious psychoses such as *manic depressive* or *schizophrenic reactions* and in organic delirium They are absent in the neuroses

Illusion —Illusions are perceptual misinterpretations Objects are perceived not as they are but in a distorted and changed form Even in normal people on tense occasions a perception may be distorted and any figure appears to be a loved one eagerly awaited or a feared person observed with dread In normal people cortical judgment and other senses quickly correct the misinterpretations

The elements likely to lead to illusions are deeply intense affective states ardent wishes or strongly urgent drives and impulses Illusions occur in *toxic states delina* and in the *psychoses*

CONSCIOUSNESS

Consciousness represents that part of the mental life of which an individual is aware at any given time It constitutes a much smaller part of our mental life than is usually recognized By some it is considered almost infinitely small as compared to the preconscious and the unconscious

Absence of Consciousness —Loss of consciousness usually fleeting is seen in *hysterical attacks* in *syncope* and following *head trauma*

Clouding of Consciousness —Clouding of consciousness is a state in which the environment is not clearly perceived The patient is in a mental fog which may be very difficult to pierce Sensory stimuli which ordinarily would call forth a response fail of apprehension Clouding of consciousness may become so pronounced as to develop into somnolence or even stupor The condition is found commonly in association with long debilitating physical illness and may be seen in functional disorders such as *hysteria*

Confusion.—Confusion is characterized by bewilderment perplexity impairment of the sensorium difficulty of grasp disorientation or poverty of ideas It occurs largely in acute mental conditions of toxic infectious or traumatic nature in *manic depressive psychoses* and *schizophrenia*

Delirium —Delirium is the syndrome characterized by restlessness clouding of consciousness disorientation visual and auditory hallucinations confusion dream like thinking and illusions It may be accompanied by anxiety and uncertainty It is usually associated with *toxic infectious illness* and is commonly of short duration On recovery there remains no memory or only patchy remembrance of what transpired

Low grade delina are often overlooked particularly in *post operative states* In these there is relatively slight affective disturbance and the patient responds to the stimulus of the doctor s presence by rousing himself to momentary clarity Similar states are occasionally found in *hysteria*

Depressive trends in deliria often end in *suicide* *paranoid delina* may progress to permanent psychotic states

DIFFERENTIAL DIAGNOSIS OF

States of Coma and Unconsciousness

Coma and unconsciousness are characterized by impairment of the capacities to receive or respond to stimuli In profound states reflexes are absent and sphincteric control is lost. Coma may develop suddenly or it may follow gradually upon increasing drowsiness and somnolence

CAUSE	DIAGNOSTIC FEATURES
Cardiovascular Disorders (Generalized)	Forward and backward failure (pp 920 941) Acute coronary insufficiency and thrombosis (p 983) Complete heart block (p. 879) Hypertensive toxemia of pregnancy (p 2638) and malignant hypertension (p 916) Define physiologic derangement for therapeutic indications
Drugs Poisons and Physical Agents	Alcoholism Sedatives and hypnotics particularly barbiturates chloral and bromides Opiates general anesthetics and overdosage with insulin (p 1241) Encephalopathies of arsenical and sulfonamide poisoning Asphyxia with carbon monoxide and other poisonous gases Sunstroke and exposure—particularly freezing
Infection (General)	Nonspecific terminal event in overwhelming infections Particularly in weakened and debilitated patients infancy and old age
Metabolic Disturbances	Profound cachexias associated with chronic disease and malignancy Hypoglycemia and diabetic acidosis Uremia azotemia and renal insufficiency Cholemia as in acute yellow atrophy of the liver Thyrotoxic crises and adrenal cortical deficiency Get urinalysis and blood chemical studies (pp 3066 3712)
Neurogenic	Increased intracranial tension from trauma, fracture of skull brain tumor brain abscess or hydrocephalus Encephalitides meningitides poliomyelitis or general paresis Cerebro-vascular accidents such as hemorrhage embolization and thrombosis Epilepsy Supplement neurologic status with spinal fluid examinations radiography electro-encephalography and specialist consultation
Psychogenic	Hysteria (p 1353) schizophrenia (p 1364) and manic-depressive states (p 1368)
Traumatic	Open and closed injuries of the skull Fractures and internal hemorrhage particularly of middle meningeal artery (p 1453) Severe burns Massive exsanguination from any portion of the body

Dream States—Dream states are periods of consciousness in which perception of environment is dulled and confused and in which hallucinatory

experiences are common On restoration of normal consciousness the patient has little or no recollection of what has transpired and may often volunteer the statement It was like a dream These disturbances may merge with delıria fugue states or dissociation of personality They occur in affective disorders such as *hysteria* and in *epilepsy*

Stupor—The term stupor as used in psychiatry may imply (*1*) a state of unconsciousness usually due to *organic conditions* in which speech and motor reactions are absent or (*2*) a seeming state of unconsciousness in which thought *is* active intellectual and sensorial clarity are present but the patient remains mute and motor activity is suspended The latter is psychogenic and seen in *depressions* and *hysteria* In this sense it is used almost synonymously with *mutism*

Catatonic stupor is a variant of schizophrenia characterized by catatonia mutism negativism hypersuggestibility and catalepsy

Coma—Coma is total loss of consciousness with absence of all voluntary motion

Emergency Treatment—The treatment of coma cannot be carried out with intelligence until the causative factor is elucidated Meantime emergency measures are practiced *for* the preservation of life as summarized below

1 Control obvious hemorrhage
2 Clear airway and administer artificial respiration (p *3766*) If necessary send for pulmotor
3 Transport to hospital as soon as condition permits
4 If blood pressure is excessive prepare for phlebotomy (p *3780*)
5 If blood pressure is at shock level bandage three extremities and set up intravenous *drip* with dextrose and saline plasma or whole blood as indicated (p 3775)
6 If urine contains acetone substitute sodium r lactate or Hartmann s solution in infusate
7 If blood sugar is low substitute 10 to 50 per cent dextrose in infusate
8 If catheterized urine contains sugar add insulin and dextrose to infusate
9 In suspected poisonings *lavage* stomach as soon as possible
10 With bitten tongue and history of *epilepsy* follow principle of skilful neglect
11 With evidences of congestive failure consider digitalization (p 858) and phlebotomy (p *3780*)
12 In suspected adrenal cortical deficiency add hypertonic saline to infusate
13 In thyrotoxic crises add iodide to infusate
14 With impaired respiration cyanosis and backward or forward fail use prepare for oxygen therapy (p 3827)
15 If spinal fluid is under increased tension bloody or xanthochromic summon the neurosurgeon after obtaining radiographs of the skull (p 1450) Examine fundi for evidences of papilledema (p 1578)
16 If spinal fluid is turbid temperature is elevated or leukocytosis is

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Dream States —Dream states are periods of consciousness in which perception of environment is dulled and confused and in which hallucinatory

Hypochondriasis —Hypochondriasis is a state of morbid anxiety about health and is usually associated with a false belief that an organ is organically diseased It may be met in *depressed states schizophrenia involutional melancholia* and other psychoses It appears also as a *neurotic manifestation* in terms of a profound unconscious drive such as narcissistic preoccupation or masochistic expiation of guilt

The difference between hypochondria is as it occurs in the psychoses and that which is encountered in the neuroses lies in the comparative severity of the underlying disturbance rather than any qualitative difference in the unconscious mechanisms Hypochondriacal preoccupation may occur in a psychotic setting or in a simple neurosis If the disturbance is particularly rigid if there is any tendency to systematization or the hypochondriacal notions become queer and distorted a schizophrenic or paranoic psychosis should be suspected

Ideas of Reference —Ideas of reference are morbid ideas that actions words and mannerisms are directed or referred to oneself they occur in *schizophrenia* and *paranoia* and may be as obstinate as delusions

Overdetermined Ideas —Overdetermined ideas are those which have multiple determinants They are anchored to many aspects of the patient's life and personality and are particularly resistant to treatment

Trend — Trend in psychiatry refers to the ideational content of any clinical syndrome A patient is said to have a homosexual trend not by virtue of his actions and feelings but by virtue of a constellation of ideas relating to homosexuality (Hinsie and Shatzky)

Progression of Thought

Progression of thought normally flows in logical sequence toward a definite conscious end There should be in the stream of thought an orderly sequence of more or less obviously related ideas passing uninterruptedly and without digression from an initial to a goal idea

Blocking —Blocking is a disturbance in the flow of thought in which expression and progression of thought suddenly cease Blocking is due to the activity or revival of a thought or complex with a powerful unpleasant affect It may result in a total obstruction of speech In some degree blocking is a universal phenomenon When it becomes a predominant obstacle to thought it characterizes *schizophrenia*

Circumstantiality —Circumstantiality is a disturbance in the flow of thought in which the goal idea is reached only after relating a mass of irrelevant and unimportant details and incidents It is characteristic of persons who cannot distinguish essentials from non essentials and who cannot maintain a sharply focused concept of what it is they wish to say It is found most commonly in the *feebleminded epileptics senile psychotics* and in *manic states*

Clang Association —Clang association is an association based on the similarity of the sound of words with no regard to the dissimilarity of ideas It is found in flight of ideas

Flight of Ideas —Flight of ideas is a disturbance of the train of thought in which the goal idea is never reached because of frequent digressions related or not It is as though thinking went on too quickly and no idea was ever completed The association of ideas may be through a chance

pronounced inaugurate massive anti biotic therapy by intra venous and intrathecal injections of penicillin (p 106)

17 Even if breath gives evidence of ingestion of alcohol remember that the intoxicated patient is subject to the same disturbances as his sober fellow citizen furthermore the drink may have been given for the relief of the very symptoms which later terminated in coma

Attention

Attention is the application of energy in the sphere of consciousness by the individual aware of the application of energy One should not speak of attention when the mind functions without the overt knowledge of the subject (Hinsie and Shatzky)

Aprosexia —Aprosexia is the inability to maintain attention It may be due to preoccupation with one s own thoughts and problems or may exist as a form of negativism Aprosexia may be selective for certain subjects

Blunting of Attention —Blunting of attention is an extreme form of in attention It is usually found in *stupor* and *coma* when painful stimuli fail to arouse the patient s attention

Distractibility —Distractibility is a state in which attention passes rapidly from one subject to another and the patient gives notice to every passing stimulus It occurs in *mania delirια* and *flight of ideas*

Fluctuation of Attention —Fluctuation of attention is the condition in which the attention varies more easily and to a greater extent than normal It is met in *organic syndromes* and *delíria*

Rational Thought (Production of Thought)

Rational thought is that thought directed by, and taking place in conscious awareness

Autism —Autism is characterized by the fact that it is uncontrolled by conscious awareness and experience it rarely leads to action and fails to take the facts of reality into consideration It is most marked in *schizophrenia* where mental activity may be completely independent of the ordinary realities of life

Delusions —Delusions are false beliefs which are not susceptible to the ordinary processes of logic or reasoning which are out of keeping with a person s education and background and which arise in response to definite unconscious emotional needs of the patient In other words the difficulty in the delusional patient is not really as it might seem in the field of thinking but in the emotional life Delusions are of many types such as delusions of persecution of grandeur of self accusation or of poverty

The delusions are reactions to highly painful affective or instinctual demands with which the patient is unable to deal adequately They may be compensatory for a painful failure or a profound sense of inadequacy or inferiority they may be projections of unconscious instinctual demands which the patient cannot tolerate because of a great sense of guilt Delusions are encountered in many mental disorders such as *schizophrenia* and *paranoia*

amnesia usually develops which may involve memory for events near the time of the treatment

Hysterical amnesia may relate to a circumscribed series of events in the patient's life usually associated with severe emotional conflict It may involve memory for all events up to a relatively recent (traumatic) point as in the *war hysterias*

Retrograde Amnesia —Retrograde amnesia involves events antedating the onset of the amnesia proper It may be seen in *head injuries* and *epilepsy* or it may be psychogenic

Anterograde Amnesia —Anterograde amnesia includes material subsequent to the onset of amnesia It is common in *senile psychoses* and after head injuries

Confabulation —Confabulation is the process by which the patient fills in gaps in his memory by falsification which he in turn accepts as actual The confabulations are easily suggested and may be directed by the person to whom they are narrated They are found in *Korsakoff's syndrome* and the *senile psychoses*

Deja Vu —Deja vu is the illusion of memory in which there is a feeling of familiarity on seeing a new object or scene of which there has been no previous observation Likewise there may be the feeling that a new experience has been encountered before

Jamais Vu —Jamais vu is the phenomenon in which familiar objects or experiences occasion the illusory feeling of newness or unfamiliarity Both deja and jamais vu are seen in *normal states* as well as in *epilepsy acute intoxication schizophrenia* and *hysteria*

Hypermnesia —Hypermnesia is an exaggerated activity of memory seen in excited states especially *manic reaction* and occasionally in *paranoia* It is usually limited to certain periods and events which are recalled with great vividness and intensity and in extraordinary detail It can be induced under *hypnosis*

Paramnesia —Paramnesia is falsification of memory and includes confabulation and retrospective *falsification* It is observed commonly in *Korsakoff's syndrome* and the *senile psychoses*

Retrospective Falsification —Retrospective falsification and illusions of memory are created by the patient to satisfy unconscious needs to embroider or distort the truth or accuracy of memory In the *paranoid psychoses* they may be used to create supporting evidence for a delusion

PERSONALITY

Personality represents the habitual pattern of behavior of the individual in terms of physical and mental activities and attitudes particularly as these have social connotations (Healy Bronner and Bowers)

Depersonalization —Depersonalization is the feeling that one is changed from what one previously was and that one's own personality has lost its identity The patient feels as though he were dead or that there is no world in which he truly exists Reality may lose its character and the environment assumes an unreal quality Depersonalization phenomena are found in *schizophrenia* and as infrequent manifestations of a profound *neurosis*

Dissociation (Double Personality) —Dissociation is the phenomenon in

stimulus from the environment through irrelevant memories or through clang association It is found in states of *manic excitement*

Neologisms —Neologisms are words or phrases coined by the patient They are frequently condensations of several words or symbolizations of unconscious material and are found in *schizophrenia*

Obsessive Thinking —An obsession is a persistently recurring and distressing thought upon which attention must be focused even though it is adequately evaluated by the intellect and stopped by the will (Maslow Mittleman) An obsession is not consciously influenced by logic reasoning or judgment

Perseveration —Perseveration is an organic phenomenon in which an echoing lag makes it impossible for the patient to keep up with changing topics An *arteriosclerotic* for example may say correctly that he is fifty six years old and then answer that he was married fifty six years and was born in 1856

Retardation —The slowing of the initiation and movement of thought is termed 'retardation The patient may complain that he thinks and speaks more slowly It is seen characteristically in the *depressed* and in *schizophrenics* during phases of depression

ORIENTATION

Orientation is an awareness of relationship to place time and persons

Disorientation —Disorientation is the inability to estimate correctly the time place or persons in an environment It may occur in any mental disease in which there is extensive impairment of memory attention or perception It is commonly found in *acute toxic states* and *organic psychoses*

JUDGMENT

Judgment is the ability to recognize the true relation of ideas and appraise their relative merits importance and significance

Lack of Insight —Insight indicates an awareness of illness and an appreciation of underlying mechanisms Lack of insight occurs in *hypochondria hysteria* and the *psychotic states*

MEMORY

Memory is the process whereby data acquired and presented to consciousness are stored later to be summoned and again presented to consciousness Even the simplest act of memory involves fixation of an impression its consideration and its subsequent recall

Agnosia —Agnosia in psychiatry is loss or disuse of knowledge of objects It is caused by emotional forces and is not associated with loss of sensation or mental deterioration

Amnesia —Amnesia is absence of memory It may be complete or partial, continuous periodic or circumscribed organic or functional

Characteristic of *senile deterioration* or *dementia* is the failure of memory for recent events with retention of memory for remote events This may progress until almost complete loss of memory is encountered In *cerebral arteriosclerosis* a more patchy but similar loss of memory is found

After convulsive therapy especially when *electric shock* is utilized an

us fears what cannot be consciously acknowledged or what is desired but which bears too great a burden of guilt for conscious recognition

Anxiety is used in contradistinction to *apprehension* which is an anticipatory dread of something that may be about to happen

Phobias —Phobias are morbid fears precipitated by the presence of some harmless or indifferent object or situation The fear arises from factors deep in the patient s unconscious the objects which explode the fear are not what is actually dreaded but are substitutes or symbols for ideas which would be even more painful In reality the fear arises from unconscious forces within the patient s mind and these are merely represented by the external object or situation which precipitates the terror

Phobias are encountered in the *neuroses* especially *psychasthenia* They may exist for anything A list of some of the more common phobias is appended

Acaraphobia —The fear that mites or minute worms infest the skin
Acrophobia —Morbid fear of being on high places or aloft in the air
Agoraphobia —Morbid anxiety when in open spaces
Anthropophobia —Fear of men
Autophobia —Morbid fear of being alone
Cancerophobia —Morbid fear of having cancer
Claustrophobia —Morbid anxiety when in narrow or enclosed spaces
Coprophobia —Morbid repugnance to filth as in defecation
Eryth ophobia —Morbid fear of blush ng or of exhibiting diffidence or embarrassment
Galeophobia —Morbid f ar of cats
Goiterophobia —Fear of goiter
Gynephobia —Fear of women or avers on to their company
Hemophobia —Morbid dread of seeing blood Illness such as fainting at the sight of blood
Mysophobia —Morbid fear of contamination or d rt
Necrophobia —Morbid fear of dead bod es
Nyctophobia —Morbid fear of darkness
Ochlophobia —Morb d fear of bei g in crowds or groups of people or in crowded places
Phobophobia —Fear of fear
Pyrophobia —Morbid anxiety about fi e
Scotophob a —Mo bi l dread of darkness (blindness)
Syphilophobia —Morbid dread of contracting syphilis Delusional belief of having the disease
Xenophobia —Anxiety or fear in presence of strangers
Zoophob a —Morbid fear of animals especially domestic animals
See also *Ph les* (p 130)

Apathy —Emotional dulling and inadequate emotional reactions to situations ordinarily giving pleasure or pain characterize apathy Apathy may result in total disinterest in the environment or the patient s own condition and lack of contact with reality

Cyclothymia —Cyclothymia refers to fluctuations characterized by alternation of optimism and pessimism cheerfulness and sadness pep and the blues In these the mood is not adequately motivated in reality and cyclothymia reaches its full development in the *manic depressive psychosis*

Depression —Defection of mind varying from feeling blue to utter despondency is depression It may be considered pathological when it is unoccasioned by external circumstances or when it is too profound or long lasting in relation to the nominal cause

Euphoria Elation Exaltation Ecstasy —*Euphoria* is an exaggerated feeling

which part of the personality becomes split off and is not available to conscious control Relatively simple examples are seen in somnambulism automatic behavior or fugues

Dissociation means many things to many people The term has been used for the vertical splits which characterize multiple personalities (Dr *Jekyll and Mr Hyde) It has also been used to denote* dissociation of different aspects of psychological functions as the dissociation between thinking and feeling which Bleuler believed the underlying disturbance in schizophrenia, it has been used for many other segmentations of mental processes such as are encountered in psychosomatic phenomena hysterical amnesia or hysterical palsy

Perhaps the best general significance to apply to dissociation would be that of any tendency to split up or fragment any aspect of the psychological functions whether the fragmentation be in a horizontal or vertical layer or piece by piece

AFFECT

Affect is ordinarily used as a synonym for emotion though it has much more definite implications of special importance in psychiatry From that viewpoint it is best defined as a feeling tone a pain pleasure accompaniment of an idea or mental representation (Healy Bronner and Bowers)

The affect (emotion) normally varies with the external environment the state of health and the position of the individual in reality The normal person s mood is expected to reflect *the total situation* In times of stress or following a great loss a mood of depression may be considered quite normal Following a high achievement a recent promotion or on a festive occasion elation normally is expected The reaction should be fitting and commensurate to the stimulus producing it neither too profound nor too lasting *Charged stimuli* are those capable of affect productions

Abnormal disturbances in affect are those motivated by forces of which the patient is unconscious and which are beyond the control of intelligence reason and conscious deliberation

Agitation—Agitation which occurs mainly in patients suffering from certain forms of *depression* or *involutional melancholia* is a state of increased activity It may range from mild restlessness to incessant over activity usually in motor behavior

Ambivalence—Ambivalence is a state of conflict in which opposite feelings are noted toward the same person at the same time as love and hatred of a parent by a child The individual may be conscious or unconscious *of the feelings and hence of the conflicts growing from them*

Anxiety—Anxiety is fear in the absence of actual danger *i e* fear occasioned by inner psychic forces of which the person is unconscious It is associated with a feeling of dread which may be related to a definite circumstance such as illness or may be entirely beyond the patient s capacity to define (I do not know myself what it is I am afraid of) It is often accompanied by the physical manifestations such as tachycardia pallor and sweating

The deep underlying tides of unconscious terror are causative forces in the production of neuroses and are to be differentiated from symptomatic fear Fear in the psychological sense represents a defense Each of

us fears what cannot be consciously acknowledged or what is desired but which bears too great a burden of guilt for conscious recognition

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Phobias—Phobias are morbid fears precipitated by the presence of some harmless or indifferent object or situation The fear arises from factors deep in the patient s unconscious the objects which explode the fear are not what is actually dreaded but are substitutes or symbols for ideas which would be even more painful In reality the fear arises from unconscious forces within the patient s mind and these are merely represented by the external object or situation which precipitates the terror

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See also *Philias* (p 1309)

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of well being often in sharp contrast to the reality situation It is a milder expression of the types of enjoyable feeling, confidence and sense of well being and capacity known as *elation* The latter may be very transient and quickly followed by an angry irritation A still more intense expression of these feelings is found in *exaltation* where the attitude is that of grandeur and all the reactions are exaggerated to the extreme A related but somewhat different disturbance of mood is found in *ecstasy* Here a quiet sense of peace and well being is practically always associated with deep religious conviction The patient feels beyond (really above) mundane activities and the difficulties of man as though he had been reborn

'Philes —'Philes represent unusual attachments beyond reason The necrophile is inordinately concerned with death, the bibliophile with books The list of the phobias may be paralleled by the philes

Panic — Panic is not merely a high degree of fear, but a fear based on prolonged tension with a sudden climax which is characterized by extreme *insecurity suspiciousness and a tendency to projection* and disorganization" (Diethelm) Severe paroxysms of panic are prognostically more serious than anxieties and may herald an approaching schizophrenic illness For instance homosexual tendencies which give rise to long standing conflict and tension not infrequently result in panic

Tension —Tension like anxiety and fear may be conscious or unconscious It is a state of mind induced by conflict and is characteristic of the *neuroses* The struggles often entirely unconscious produce a feeling of *strain* and tautness apparent in the ' tense facial *expression* A *tense* person is tight fidgety restless tremulous a chain smoker, and the like

Appetite

Appetite is a natural longing or desire it usually refers to food but may be applied to learning sex or music

Anorexia —Anorexia is loss of appetite A distinction is made between true anorexia nervosa and the anorexia due to distorted ideas about the stomach and intestinal tract or delusions about food See p 1768

Bulimia —Bulimia is excessive insatiable appetite It is encountered as a compulsive manifestation and may occur after head injuries or encephalitis as well as in *hysteria* Obstinate bulimia may lead to obesity (p 695)

Morbid Hunger —Morbid desires for food are found in various emotional and mental disorders, such as *anxiety states compulsion neuroses* and *schizophrenia*

Eating may be utilized as an instrument to allay anxiety and tension and in response to a craving that is only vaguely associated in consciousness with ordinary hunger

Sexual Behavior

Normal sexual function in the adult includes much more than the genital aspects of intercourse A well integrated sexual relationship embraces genuine feelings of love for a partner of the opposite sex the desire to engage in the sexual act with that person the capacity to perform the act with resultant orgasm for each a post coital feeling of release and gratification and a capacity to relax and fall asleep There should be adequate awareness of the partner s moods and sexual needs feelings of ten

derness the necessary preliminary foreplay without revulsion or fright and a capacity for release in action and language The estimate of what constitutes a normal and adequate sexual relationship is complicated by social economic and emotional factors as well as conscious and unconscious attitudes It can be estimated only by a thorough investigation of sexual functioning and total personality

A man may be genitally adequate and capable of erection and orgasm and yet not be healthy sexually he may be unable to form any attachment for a woman he may need to use prostitutes exclusively he may need to escape immediately after completion of the sexual act intercourse may be possible only after drinking or there may be post-coital restlessness and irritability

Women have parallel difficulties they may be incapable of ardor or orgasm the climax may be premature or delayed they may be inconstant in their male attachments or they may require masturbation prior to intercourse or preliminary alcoholism Any of these manifestations of emotional difficulty point to a profound unconscious conflict which may produce evidences in a host of ways seemingly unrelated to the sexual life

Exhibitionism —Exhibitionism is the need to display the body its parts or sexual activities in order to arouse sexual interest or to compensate for unconscious feelings of sexual inadequacy or impotence

Fetishism —Fetishism is erotic gratification through the use of a fetish such as an article of clothing or some nongenital portion of the body The object arouses erotic impulses usually through unconscious associations

Homosexuality —Homosexuality is the love for a person of the same sex In the form of interest as in friendship it is a component of every individual s sexual life It is considered noteworthy or pathological when it exists in the place of heterosexual interests and love and when it becomes the only obligatory compulsive avenue of sexual expression

Incest —Incest is a sexual relationship between close blood relations (brother and sister parent and child) The discovery of the presence of unconscious incestuous desires in all humans is one of the most important results of psychoanalytic research Incest between siblings is far from rare in childhood and adolescence

Nymphomania —Nymphomania a *compulsion neurosis* focussed on genital activity consists of an excessive insatiable impulse in women to heterosexuality

Perversions —Perversions are sexual practices which markedly deviate from the so called normal or average Previously the subject of perversions vastly preoccupied psychiatrists but as psychoanalytic research has widened knowledge of sexual behavior the use of the term is more restricted and the concept less emphasized Many practices formerly looked on as perverse are now understood to be concomitants of normal sexual intercourse especially the milder variants employed in sexual foreplay Perverse tendencies become perverse only when they compulsively dominate sexual behavior to the exclusion of normal activity

Satyriasis —Satyriasis the male equivalent of nymphomania is excessive insatiable heterosexual needs Although the excessive activity occurs in the sexual sphere the drive which actuates the satyrism need not necessarily be erotic despite its cloak

Voyeurism —Voyeurism or peeping is sexual pleasure obtained by looking at the genitals or genital activities of another The looking is itself the ultimate aim of the sexual act and the act is usually committed secretly

Masochism —Masochism is that condition in which sexual satisfaction depends on the subject himself 'suffering pain ill treatment and humiliation" (Freud) It is characterized by the conscious or unconscious need to experience pain to achieve full sexual gratification

Sadism —*Sadism is the accomplishment of sexual gratification* from the infliction of pain on others It may vary from overtly perverse behavior as in whipping to the unconscious sadistic component of erotic practices as in humiliating or ill treating the love object A mild sadistic element is not infrequently present in normal love making as gentle biting

Sexual Function

Ejaculatio Praecox —Ejaculatio praecox is a condition in which ejaculation and detumescence occur before or too soon following entrance to permit satisfactory intercourse for man or woman It is a common *neurotic disturbance* of sexual function (p 2408)

Ejaculatio Retardata —Ejaculatio retardata is the relatively rare inability to achieve orgasm despite prolonged intercourse It may result in *painful intercourse with swelling and irritation of the penis* Mild degrees are not rare and often alternate with ejaculatio praecox

Frigidity —Frigidity is the inability of the female to achieve orgasm despite the genital adequacy of the male Although commonly used to refer to genital failure it also applies to absence of sexual feelings in general It may disappear and re appear under special circumstances

Impotence —Impotence is interference with the male capacity to have sexual intercourse the inability to have and maintain an erection It may be relative or absolute organic or psychogenic, it may vary with different women and under different circumstances A man may be potent with a prostitute and impotent with a woman for whom he has feelings of love he may be potent at the beginning of but lose his erection during intercourse The organic aspects of impotence are elsewhere described (p 2409)

Priapism —Priapism is persistent abnormal painful erection of the penis usually without accompanying conscious sexual desire The priapism may last from hours to weeks and lead to secondary changes in the corpora cavernosa It occurs in cord lesions and leukemia or it may be psychogenic In the latter instance a course of intensive psychotherapy is indicated along with the use of sedatives and relaxation baths Occasionally spinal or epidural anesthesia must be employed to obtain a brief respite

Spermatorrhea —Spermatorrhea refers to the spontaneous discharge of spermatic fluid usually without evidences of sexual desire or erection

Dyspareunia —Dyspareunia indicates painful intercourse and since it may be due to technical and organic as well as psychogenic causation it is elsewhere discussed (p 2491)

Sleep

Normal sleep is characterized by the suspension of consciousness absence of reactivity to environment increase in the thresholds of general

DIFFERENTIAL DIAGNOSIS OF

Insomnia

Without knowledge of the causation of insomnia drug therapy holds little promise First principles in management consist of efforts to remove or correct etiologic factors

CAUSE	DIAGNOSTIC FEATURES
Errors in Hygiene	Excitement, anxiety fear fatigue overheated or chilled room, bad ventilation, undue noise or light, excessively soft or hard mattress too many or too few bedclothes too many pillows insufficient activity during day or undue amount of sleep and late nap in afternoon or evening
Pain	Especially with nocturnal exacerbations as in peptic ulcer and disturbances of bone
Fever	Particularly in infections characterized by night sweats such as tuberculosis and rheumatic fever Check fever chart, sputum get skin test and sedimentation rate
Drugs	Caffeine (coffee tea and cola drinks) Alcoholism Adrenergens (amphetamine ephedrine and allied preparations) Toxic doses of thyroid extract. Idiosyncrasy to opiates
Neuroses	Particularly in anxieties (pavor nocturnus) neurasthenia and hypochondriasis
Psychoses	Schizophrenia manic-depressive episodes and general paresis Examine spinal fluid (p 3734)
Neurogenic	Particularly cerebral arteriosclerosis and encephalopathies.
Metabolic	In rickets hyperthyroidism, diabetes mellitus (polyuria) renal insufficiency (nocturia) and pruritic conditions Examine urine Obtain blood chemistry (p 3712) and B M. R. for therapeutic indications
Circulatory	With orthopnea due to backward failure (p 941) From paroxysmal cardiac irregularities (p 873) With night terrors especially in aortic disease and pericarditis (p 1007) Check physical findings with x ray and electrocardiogram.
Gastro-intestinal	Hunger pain in peptic ulcer Gastric dilatation due to retention or a meal at bed time Constipation and distention. Fermentation and diarrhea Nausea and vomiting Supplement *examinations of stool and gastric contents with* barium x rays
Respiratory	Nasal obstruction and post nasal drip Cough expectoration or dyspnea Check physical findings with x rays of sinuses and chest.
Genito-urinary	Ungratified eroticism. Nocturnal emissions Dysuria polyuria urinary retention and urinary frequency Examine urine and prostate
Pregnancy	From size of uterus or activity of fetus
Neuromuscular	Particularly with back pain and cramps in the thighs and calves

sensibility and reflex irritability, diminution of the basal metabolic rate lowering of blood pressure and a slight rise in temperature

The nature of sleep is still in dispute Pavlov believed that sleep was due to cortical inhibition There is accumulating evidence of the existence of a *sleep regulation center* in the mesencephalon the region in the periventricular gray matter of the third ventricle the neighborhood of the infundibulum or the mesial parts of the thalami In man, nocturnal sleep is regarded as normal The criterion of a satisfactory period of sleep is a feeling of rest and refreshment after awakening

Many of the popular legends about sleep have little basis in physiologic fact The ritual of the need for one long period of sleep in twenty four hours and of the necessity for remaining asleep a certain number of hours is a matter of custom more than anything else Animals sleep at frequent short periods throughout the day, a short nap may provide considerable rest and refreshment whereas a prolonged sleep may be followed by a sense of fatigue and heaviness

Insomnia —Insomnia occurs in three forms There may be inability to fall asleep but a sound sleep once sleep is achieved, there may be a tendency to waken repeatedly as the result of terror dreams throughout the night there may be no difficulty in falling asleep but early wakening with inability to return to sleep Each variety of insomnia requires different management the patient who has difficulty falling asleep should have a rapidly acting soluble drug whereas those who waken repeatedly or in the early hours of the morning do better with the insoluble barbiturates (p 3839)

Treatment of Insomnia —Insomnia is best managed by elimination of the cause The use of drugs is postponed until other measures have been exhausted

1 Correct errors in hygiene particularly relative to the bedroom
2 Discontinue naps during day or early evening
3 Walk before retiring or at least exercise before open window
4 Omit fluids after dinner in those with polyuria nocturia or frequency
5 Omit feedings after dinner in those who suffer gastric retention or flatulence
6 Give feedings of milk and crackers or soup for those with hypermotility or peptic ulcer
7 Evacuate rectum with suppository or rectal flush in those who complain of flatulence
8 Stop all stimulant drugs
9 Forbid coffee tea and cola drinks after 4 P.M
10 Reduce alcohol to total two daily drinks Suggest hot toddy at bed time
11 Advise warm tub or bed sponge before retiring for febrile with nocturnal sweats
12 Arrange pillows for orthopneic cardiacs
13 Suggest steaming at bed time for respiratory sufferers with post nasal drip or cough Avoid sprays and drops with adrenergens
14 Finally if necessary prescribe soluble hypnotic such as *seconal so*

dium 100 mg (1½ grains) for rapid action, *sodium amytal* 180 mg (3 grains) for delayed action, and *tuinal* (equal parts of seconal and amytal) 180 mg (3 grains) for continued effects

Inversion of Sleep Ratio.—Inversion of sleep ratio is a condition in which the patient sleeps by day and has insomnia at night. The nocturnal insomnia may be associated with aggressive sexual behavior and voracious eating. It is found in maladjusted young people, *chronic encephalitis* and *schizophrenia*.

Narcolepsy.—Narcolepsy is characterized by short spells of uncontrollable sleep lasting from minutes to hours. It occurs under circumstances ordinarily associated with wakefulness, as for instance at work. At times the patient is not really asleep but is in a sort of trance; he is usually fairly alert when attentiveness is demanded but slips easily into sleep. During the narcoleptic spell the patient is relaxed, his color is good, the pulse rate is slow and breathing is slow and regular. The condition may be set off by sudden emotion, either pleasurable or unpleasurable. The patient, once asleep, is easily aroused. Nocturnal sleep is usually normal.

The pathology and etiology of this condition are not known, but it is observed in *subacute encephalitis*, following *head trauma*, in *cerebral neoplasms*, especially those of the third ventricle, and associated with *epilepsy*. It is believed that the attacks are due to involvement of the mesencephalic centers or inhibition of various parts of the central nervous system.

Somnolence.—Somnolence is unnatural sleepiness or drowsiness, usually at inappropriate times such as at work, in the theater or in the classroom. It may be related to *hypothyroidism*, but most commonly is seen in the *neuroses*.

Treatment of Somnolence and Drowsiness.—The management of somnolence involves correction of hygiene and the use of metabolic and pharmacologic measures.

1. Eliminate errors in hygiene.
2. Treat nocturnal insomnia (p. 1706).
3. Prescribe low calory diet for obese (p. 449).
4. Proscribe alcohol.
5. Stop use of sedatives.
6. Give thyroid extract in conditions of hypometabolism (p. 1189).
7. Give androgen or estrogen in climacteric states (p. 275).
8. Suggest morning purge with 1 or 2 teaspoonfuls of sodium bicarbonate and two glasses of hot water on arising.
9. Provide for clearing of airway with steaming or nasal sprays.
10. Order hematinics (p. 1018) in anemias.
11. Finally, prescribe amphetamine sulfate 2.5 to 10 mg. on arising with repetition at noon if necessary.

Prolonged Sleep.—Prolonged sleep, as the name implies, is the simple prolongation of sleep from days to months. It is found most commonly in acute and chronic *encephalitis*.

Terror Dreams.—Terror dreams are those in which the anxiety attached

sensibility and reflex irritability, diminution of the basal metabolic rate lowering of blood pressure and a slight rise in temperature

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ality structure The material that has detached itself from conscious control to form a secondary personality leads the individual from the ordinary places and activities of life Thus the patient may undertake a journey and find himself in a distant city with no conscious knowledge of why he made the trip how he arranged it or any of the details

Somnambulism —Somnambulism or sleep walking is the equivalent of the fugue except that it occurs during sleep In these acts either literally or symbolically the patient attempts to act out a desire or need which he cannot permit himself while conscious

The somnambulistic act and the accompanying dreams are not usually remembered The patient unless awakened returns to bed and resumes usual sleep Occasionally the somnambulist meets with an accident and suffers injury from a fall or from broken glass

Compulsion (Compulsive Act) —Compulsive or imperative acts are morbid irresistible urges to perform apparently useless and meaningless rituals They may be simple or complex They may afford unconscious gratification or protection to the patient or they may represent a substitute expression for the gratification of a repressed desire or experience

Repetitive Acts —Repetitive acts are forms of compulsive behavior in which the patient repeats simple acts such as counting the books on a shelf or the boards in the floor They may become complicated and burdensome as in many compulsive ceremonials The necessity of avoiding or renouncing these rituals may be accompanied by great discomfort and anxiety They are part of the syndrome of the *compulsion neuroses* (p 1345)

Echolalia and Echopraxia —*Echolalia* is the meaningless repetition of words or phrases spoken by others It is an expression of automatic obedience Instead of answering a question for instance the patient repeats it again and again *Echopraxia* is the meaningless repetitive imitation of acts or movements performed by others in the patient s environment These phenomena are found in *schizophrenia*

Mania —Mania is a state characterized by exaggeration of ideas feelings and motor activity See *Manic Depressive Psychosis* (p 1368)

Hypomania —Hypomania is a less intense form of mania

Hypermania —Hypermania is a more intense form of delirious mania

Mannerisms —Mannerisms are gestures or other forms of expression characteristic of an individual In psychiatric patients mannerisms may become disproportionately important and acquire the significance of symptoms

Negativism —Negativism is opposite (negative) behavior or speech The patient characteristically does or says the opposite of what he is asked or expected to do This type of behavior is seen in *schizophrenia*

Paralalia (Speech) —Paralalia usually refers to any speech defect but may indicate the speech defect characterized by the substitution of one letter for another

Psychomotor Activity —Psychomotor activity is movement that is psychically determined the action resulting from an idea or perception It may be exaggerated (overactivity) diminished (underactivity) or disjointed (disactivity)

Stereotypy —Stereotypy is the constant seemingly meaningless repe-

to the content of the dream is so intense as to awaken the patient with tachycardia sweating restlessness and breathlessness

Hypnotic Sleep —Sleep induced through hypnosis is usually for diagnostic purpose, although occasionally the induced sleep itself is used directly as a *healing factor*

DIFFERENTIAL DIAGNOSIS OF

Somnolence and Drowsiness

The problems of somnolence are similar to those of insomnia with which they may be associated Rational therapy depends on the elucidation of the causative mechanism

CAUSE	DIAGNOSTIC FEATURES
Errors in Hygiene	From overheated room or insomnia of previous night From gluttony and over indulgence in alcoholic beverages With fatigue and excessive exposure to intense cold
Pharmacologic	Sedatives hypnotics and opiates
Neuroses	Neurasthenia hypochondriasis or hysteria
Psychoses	Schizophrenia depressed stages of manic depressive disorders and general paresis Examine blood and spinal fluid.
Neurogenic	In cerebral arteriosclerosis encephalopathies and encephalitides
Metabolic	Hypothyroidism acromegaly pituitary basophilism, Frohlich s syndrome adrenal cortical insufficiency hypogonadism diabetes mellitus and uremia Get urinalysis (p 3667) blood chemistry (p 3712) and B M R. for therapeutic indications
Gastro intestinal	With obstipation and auto-intoxication
Respiratory	With apro exia especially in upper respiratory infections
Blood Disorders	Especially with anemias Get hemogram (p 3704)
Infectious	In epidemic encephalitis and trypanosomiasis

Motor Behavior

Motor behavior is judged by the neuromuscular activities of the patient in his gait speech posture expression and activities

Abasia —*Abasia inability to walk is usually associated* with *astasia* inability to stand erect

Astasia abasia —Astasia abasia is a symptom of mental conflict most commonly found in *hysterical conditions* It consists of an inability to stand or walk *and is based on abulia a loss or deficiency of will power*

Automatism —*Automatism is a condition in which activity is carried* out without conscious knowledge It is seen in *catatonic schizophrenia* and in certain *hysterical states* Tics may be considered as automatisms

Fugue —The fugue or flight is an expression of dissociation in person

CHAPTER 61

PSYCHIATRY THE ETIOLOGY, CLASSIFICATION AND DIAGNOSIS OF MENTAL DISEASES

THE ETIOLOGY OF MENTAL DISEASE

THE mechanisms by which the nerve cells and tissues become deranged do not differ from those that are operative in the other organs of the body Cellular injury and death result from physical and chemical causes from trauma from the invasion of neoplastic tissue from the effects of infectious processes (particularly syphilis) from nutritive disorders arising from vascular disease and occlusion and from poisonings as the result of the influences of drugs or chemicals arising endogenously in the course of disturbed metabolic processes or of exogenous origin in occupation or criminal poisoning

Beyond these tangible and often demonstrable mechanisms there is a vast reservoir of mental disease in which neuroses and psychoses and disturbances in personality and character arise without demonstrable exogenous cause and in which there are no clearcut pathologic changes These states are obviously disorders of a functional nature often arising as the result of derangements in cerebration or the emotional development of the individual

Organic Disease.—The histopathologic forms of mental disease include the *congenital abnormalities* such as mental deficiency the *infectious states* particularly syphilis the *traumatic and post traumatic afflictions* the *vascular group* secondary to arteriosclerosis the *psychoses associated with neoplastic disease* and *miscellaneous neurologic disorders* such as multiple sclerosis and paralysis agitans Afflictions in these categories are essentially secondary manifestations of more fundamental tissue derangements and management is directed toward the correction or elimination of the noxious origin See p 1374

Toxic or Metabolic Derangements.—Mental diseases due to toxic or metabolic derangements are rarely associated with demonstrable histologic change but the offending substance may often be suspected or demon

tition of any act The maintenance over long periods of a fixed posture or place has also been called stereotypy It is seen in *schizophrenia*

Tics —Tics are involuntary movements of certain muscle groups The resultant motion resembles an organized action in contrast to other in voluntary motions Tics may be organically induced or psychogenic the latter sometimes being spoken of as habit tics

Verbigeration —Verbigeration is the meaningless repetition of sentences phrases or words It is stereotypy as expressed in language and is encountered in *schizophrenia*

SPEECH

The accomplishment of speech is an attribute almost wholly limited to human beings It is a highly complex neuromuscular activity

Aphonia —Aphonia indicates loss of phonation although articulation is preserved The patient talks in a whisper It is most commonly found in *hysteria* but it may be due to bilateral paralysis of the vocal cords or disease of the larynx (*q v*)

Mutism —Mutism is the state of being dumb silent or voiceless in the absence of structural changes (deaf mutism) It results from psychic causes and is commonly associated with negativism It represents a refusal to talk and is seen in *schizophrenia depressions involutional melancholia* and *hysteria*

Stuttering Stammering —Although some specialists in the disorders of speech distinguish between stammering and stuttering in general usage the latter is considered a more violent form of the former Each is characterized by spasmodic utterances and blocking breaks and repetitions usually associated with sputtering The difficulty may be constant or appear only under emotional stress Occasionally stutterers can sing without noticeable difficulty

There is some suggestion that stuttering is organic and that it results from emotional factors exerting their influences on a definite nucleus Often it is hereditary and may be related to handedness or dominance

POSTURE

Cataplexy —Cataplexy consists of paroxysmal attacks induced by emotional excitement such as intense laughter or irritation in which there is postural collapse of the whole body with loss of muscle tone The person sinks to the ground consciousness may or may not be lost but the deep reflexes are abolished Cataplexy is usually grouped with *narcolepsy* as a variant of the paroxysmal disorders The cause is not *definitely* known

Cereaflexibility —Cereaflexibility is the wax like flexibility and immobility of the extremities met in catatonic stupors The extremities placed in any awkward uncomfortable position are maintained for abnormally long periods of time

Catalepsy —Catalepsy is usually used synonymously with flexibilitas cerea It may refer to any form of sustained immobility These states are most common in *schizophrenia* and *hysteria*

This apologetic introduction is planned to placate erudite specialists who may be assured that every effort has been made to avoid the perpetration of excessive violence to current psychological and psychiatric concepts It is the present plan to subdivide mental disease into (1) those conditions characterized by *mental deficiency* which are measurable through intelligence quotients the less tangible but by no means less real (2) *neuroses* (3) *idiopathic psychoses* and (4) *symptomatic psychoses*

Mental Deficiency —The conditions of idiocy imbecility and moronity are discussed in Chapter 63 (p 1332)

Definition of Neurosis —The neurosis is a psychological disturbance that arises as a reaction to stress It is unaccompanied by any serious alterations in the evaluation of reality It is a *partial reaction* in that the personality is otherwise well integrated and socially organized It is more common and usually less severe and disabling than the psychosis

Definition of Psychosis —The psychosis is a total personality reaction that is disabling and severe The personality is distorted and disorganized and the appreciation of social and sensory reality is entirely destroyed

TABLE 86 —FACTORS DIFFERENTIATING NEUROSIS FROM PSYCHOSIS

Neurosis	Psychosis
Partial reaction	Total reaction
Less severe	Disabling and more severe
Anxiety and compulsion behavior of thoughts	Hallucination and delusion
Personality remains socially organized	Personality distorted and disorganized
Appreciation of social and sensory reality seriously affected	Appreciation of social and sensory reality entirely destroyed
No great interference with reality testing grasp of social relationship and appreciation of external world not seriously compromised	Grasp of social relationship and appreciation of external world greatly compromised

and often replaced by hallucination and delusion The grasp of social relationships and the appreciation of the external world situation are greatly compromised and the afflicted patient is greatly incapacitated in so far as normal interrelationships are concerned

Distinction Between Neurosis and Psychosis —The differential diagnosis between the neurosis and the psychosis is of utmost importance in the arrangement of a therapeutic program and in prognosis It is summarized in Table 86

Idiopathic Psychoses —The idiopathic psychoses are those in which there is no demonstrable etiologic agent The most important of these baffling and disturbing clinical entities are schizophrenia (p 1364) and manic depressive insanity (p 1368)

Symptomatic Psychoses —The symptomatic psychoses are those in which the mental aberration complicates a more fundamental anatomic or functional disturbance The commoner etiologic factors are trauma fever infection neoplasm cerebral vascular disease metabolic derangements poisonings and neurogenic disorders

strated *Endogenous* metabolic causes include fever acidosis azotemia, hypoglycemia hyperglycemia avitaminosis and endocrinopathies *Exogenous* toxic agents which produce mental disease, include alcoholism saturation of the nervous system with metals particularly lead mercury and arsenic, and the chronic use of opium derivatives bromides cocaine and barbiturates See p 3839

Functional Disturbances —The mental disorders that arise without tangible histopathologic chemical or metabolic causation are more difficult of definition classification and treatment Since these afflictions particularly the neuroses constitute the vast reservoir of mental disease seen in clinical practice they offer a real challenge to the practitioner who is required to have some concept of their nature if he is successfully to deal with them

Dynamic Psychology —Students of dynamic psychology explain functional mental afflictions in terms of the *conflicts* which inevitably result from the inability of the individual to adjust himself to his environment and his society In our civilization each of us is subject to disappointment frustration stress and anxiety There is necessarily some measure of emotional conflict which must influence and sometimes cause mental illness Our present civilization allows for many of us highly inadequate expressions of our needs We are compelled to curb desires school ourselves in disappointment and wait upon the achievement of ambitions We must regulate our behavior repress our emotions and wishes and fit our individual expressions to the environment and society in which we live The necessary compromises may be reflected by the production of neurotic behavior psychosomatic disturbances or actual psychoses

Freudianism —The monumental contribution of Sigmund Freud seems best adapted to function as a working hypothesis for the clearer understanding and more intelligent management of the common neuroses as seen in private practice This statement is not to be interpreted as a wholehearted and complete acceptance of all the theories of Freud, nor is it to be regarded as indicating that the practitioner should become a psychoanalyst or necessarily refer his patients to the accredited specialists for psychoanalytical therapy It is sufficient in this place to emphasize that the acceptance of the Freudian theory of the neuroses and the approval of widespread psychoanalytic therapy constitute separate and distinct discussions Freud himself has made this clear when he wrote the future will probably attribute far greater importance to psychoanalysis as the science of the unconscious than as a therapeutic procedure

THE CLASSIFICATION OF NERVOUS AND MENTAL DISORDERS

The exigencies of the general practice of medicine require that the practitioner who is concerned with all of the branches of his profession be provided with expositions of specialist fields expressed in terms sufficiently plain so that they are readily comprehended and yet not so simplified as to rob the material of its meaning With these requirements in view and with due reverence and apologies to neurological specialists and psychiatrists the following classification is tentatively presented for present usage It is intended to furnish the practitioner with an intelligent concept of mental conditions and a definitive method of managing the afflicted patient

dwells on friendships religious attitudes and concepts social and community responsibilities and reactions to authority On a more personal basis the patient is questioned as to emotional fluctuations reactions to disappointment and success attitudes toward money conscientiousness punctuality hypersensitivity irritability mood changes personal habits and the presence of phobias or compulsions

When it comes to a discussion of the present illness the patient is requested to express an opinion as to causative mechanisms situations or occurrences This viewpoint may be supplemented or corroborated by a mother father wife husband child or other member of the family preferably quizzed in the absence of the patient The acquisition of these data does not require any particular training or special aptitude though it is time consuming The whole history often cannot be obtained in one formal sitting but may be pieced together over a period of time after a number of contacts

Physical Examination —The physical examination of the psychiatric patient must be most exhaustive In addition to the routine survey a gross neurological examination is mandatory the fundus oculi must be scrutinized and a temperature record must be recorded See also p 3468

Laboratory Data —The history and physical examinations in psychiatry require supplementation by a battery of laboratory tests including routine urinalysis hemogram (p 3704) blood chemistry basal metabolic rate determination and a serologic test for syphilis (p 337) In the presence of severe or persistent disorders cerebrospinal fluid is obtained evidences of increased intracranial pressure are sought and the fluid is examined for its cytologic bacteriologic and serologic characteristics and for colloidal gold reactions (p 3737)

Neuropsychiatric Consultation —The neuropsychiatric consultant is utilized freely by the sage practitioner In highly specialized institutions reference is made to psychiatrist organic neurologist and neurosurgeon Additional examinations include radiography of the skull electroencephalography ventriculography encephalography and laminography Psychometric tests may be required (p 1395) An ophthalmologist may be called for more accurate delineation of the changes in the visual fields or the fundus oculi (p 1545) The otologist is asked to perform audiometry and vestibular tests (p 2017) In desperation it may be necessary to perform an exploratory craniotomy or laminectomy In the end the practitioner often finds himself beyond his depth in his attempt to integrate specialist findings and under these circumstances it is wise to call in a seasoned internist to act as referee (p 3890)

THE DIAGNOSIS OF MENTAL DISEASE

The diagnosis of mental disease places a severe tax and responsibility upon the practitioner After the recognition of the psychiatric component it becomes necessary to define the diagnosis in terms of mental deficiency neurosis, idiopathic psychosis or symptomatic psychosis and then seek to determine or exclude the presence of local or systemic disturbances of more fundamental etiologic significance

The practitioner owes it to the patient and himself to arrive at the diagnosis of the neurosis or the idiopathic psychosis only through the tedious methodology of exclusion Once having conducted his survey he must pave the way for re investigation at stated intervals since latent processes may reach the surface of clinical recognition after periods of months or years Exemplifying this are examples of brain tumor with herald psychiatric manifestations and later organic findings definitely not present at the initiation of the difficulty

History —The psychiatric history must be broad in scope since the sub ject matter deals with all processes which disturb the adjustment of the patient compromise his attainment of ordinary gratification deprive him of a proportionate amount of happiness and self satisfaction and limit his capacity to get along with his fellows within the social structure of the community in which he dwells The psychiatric history concerns itself with the individual in the broadest aspects of his bodily health It deals with his social marital and business adjustments moods and reactions to the problems of every day life

The psychiatric examination need not be conducted on a formal basis The patient is put at his ease and given every opportunity to dilate on his individual problems In the course of the discussion inquiry is made concerning the incidence of nervous and mental disorders alcoholism syphilis and epilepsy in the family If it is possible a birth history is ob tained particularly with reference to length of labor use of instruments and the amount and degree of asphyxia Emphasis is placed on childhood development particularly such factors as weaning cleanline s temper tan trums masturbation and thumb sucking Some idea should be obtained of early family training with particular reference to the position of the pa tient in the family circle and in relationship to grandparents parents brothers sisters and household servants Early personality traits are im portant, especially in contacts with other children The school history deals with scholastic progress disciplinary problems crushes' on school mates and teachers participation in sports and extracurricular activities and rela tive academic standing It is necessary to establish thorough information concerning psychosexual adjustments The patient is quizzed concerning masturbation homosexual manifestations whether overt or implied hetero sexual adjustment premarital marital and extramarital intercourse atti tudes toward the conjugal partner and children and deviations in sexual practices

Equally important are the histories of work and social adjustments The patient s attitude to business profession or household is noted Em ployees are questioned concerning their ability to stick to a job and take orders Employers are interrogated concerning ambitions attitudes to em ployees and competitors and their feelings of security The social history

ple mechanism of an outpouring of hatred injustice or intolerance fancied or real

There is no contraindication to the practitioner functioning as a passive psychotherapist In exchange for the time expended his patient for whom he has actually performed no active service may leave his presence assured and assuaged

Information Interpretation and Explanation —Much of the usefulness of the practitioner is related to his role as a pedagogue dealing with the problems of general hygiene Many fears are based on erroneous conceptions of physiologic facts The teachings of body hygiene dietotherapy intestinal and sexual habits and the care and feeding of children and their parents are part of the routine of private practice Expositions of these problems should be given simply and without condescension or underestimation of the nature and extent of the anxiety which misconceptions may engender The discourse should be in the vernacular avoiding technical terms which serve to confuse the auditor

As a corollary to the dissemination of information the practitioner functions *to correct misinformation* All that many patients need to be told is that old wives tales are just not so For example it is not necessary to have a bowel movement each day frightening the pregnant woman does not produce congenital malformation of the child masturbation does not result in rings under the eyes or acne vulgaris eating meat does not cause elevation of blood pressure nor the combination of bananas and water a fatal indigestion a blow on the female breast does not produce cancer any more than eating acid foods produces rheumatism venereal disease is not acquired on a toilet seat nor is pregnancy the result of bathing in a tub previously used by a younger brother The anxieties that arise from these myths can often be dissipated by statements of simple fact Persistence of the symptom despite lucid explanation serves as a *therapeutic test* and indicates that the disturbance is on a more profound level The rigidity of the neurosis then is such as to require specialist care

Reassurance —In a positive way psychotherapy is best practiced effectively when the physician gives his patient reassurance Most patients consult their physician with an *aura of anxiety* If a finger is cut there is dread of infection the presence of a lump precipitates cancerophobia and an excoriation of the penis brings fear of syphilis Frequently the patient comes to relieve his anxiety rather than to submit to therapy Reassurance that his fears are unwarranted is accordingly a powerful psychotherapeutic measure

Complete physical examination is perhaps the most potent method of providing reassurance but excessive examination with a formidable battery of tests may lead the anxious patient to assume the presence of serious illness During his investigation the practitioner must not indulge in careless or technical conversation lest the patient develop a sense of insecurity from the use of words foreign to his everyday conversation To the anxious patient a lump means cancer a murmur heart trouble absent reflexes imminent insanity infected tonsils septic poisoning

Lending Moral Support —In emergencies the physician s very presence supplies moral support which may be more beneficial than medication

CHAPTER 62

PSYCHOTHERAPY

NON-TECHNICAL PSYCHOTHERAPY

FOR THE most part except in expert hands psychotherapy is a guile less and unobtrusive exercise The practitioner indulges in it when he smiles reassuringly upon entering a sick room when he pats his patient s hand or spends the night in the home of a desperately sick patient friend hopelessly afflicted with an inoperable carcinoma when he discounts 50 to 100 mm of mercury in giving a blood pressure reading to an anxious and unstable individual, and when he prescribes a placebo for his patient and surrounds the prescription with the trappings of a potent drug Each prescription that is written each injection that is given and each proce dure that is executed is clothed with an aura of hope trust and faith This may not be the intent of the physician at all times but it is implica tion received by the patient

Notwithstanding their protestations to the contrary the technical spe cialists including the surgeons practice psychotherapy The operator is a psychotherapist when he reassures us who are on the cutting end of the scalpel that it won't hurt and when he asserts that the intended proce dure is without discomfort morbidity or mortality ' For the most part massage and physiotherapy particularly with electrical apparatus owe their beneficences to the element of suggestion

Few therapeutic modalities are more important and more potent than psychotherapy None requires such tender devotion or respect for once lost faith like virginity can never be regained

THE PRACTITIONER AS A PSYCHOTHERAPIST

The practitioner psychotherapist may serve his patient in good stead without great formal training Equipped only with patience good com mon sense and a sympathetic desire to be helpful he is capable of han dling all but the most difficult challenges

Ventilation —At times the practitioner needs only to listen sympathet ically to the recital of his patient's problems and tribulations The major ity of disturbed individuals ask for no more than an attentive ear and some intangible evidence of understanding listening Ventilation is as an cient as the religious confessional and as new as the *catharsis* of certain forms of modern analytic therapy It is a form of therapy that has great power for good and little prospect for evil It relieves tension by the sim

The practitioner should give as little advice as possible and aim to guide the patient in the working out of a solution for his individual problem It is good discipline to suggest to the patient after the facts have been gathered to sleep on the matter and then make an attempt to think things out This serves to stimulate initiative and from the practitioner s point of view terminates a protracted interview

The giving of advice has at least one other advantage in that the patient may find himself incapable of executing the suggestion however sound and appropriate As a result he develops even greater feelings of insecurity and doubt defeating the intended therapeutic purpose and erecting a barrier between advisor and advised

Play Hobbies and Recreations—The physician should have an accurate knowledge of the occupational and diversional facilities that are available in his community The cultivation of hobbies indulgence in educational and artistic opportunities exercises or sports and an acquaintance with nearby places of sanctuary and refuge constitute psychotherapeutic devices of considerable importance The ingenious physician often succeeds well in utilizing even the meager resources of his locale

Rest and Relaxation—There is no possible manner of overestimating the beneficences of rest and relaxation At times the physician accomplishes this for his patient by suggesting the interpolation of a rest period or nap after luncheon in the middle of the day or after the evening meal A housewife may be relieved of some of her household duties by requesting more cooperation and thoughtfulness from her children Reference is made elsewhere (p 3755) to the devices of the partial rest cure consisting in spending part of the day in bed or loafing for a weekend (p 3754) More elaborate devices include the complete rest cure (p 3754) using the Weir Mitchell technic (p 3754) or a modification thereof Wealthier patients gild their infirmities by taking a vacation or visiting a spa (p 3764) preferably one at a great distance

Occupational Therapy—The use of occupational therapy in the home requires only the exercise of ingenuity by patient or physician Reference is made elsewhere (p 3764) to the amazing frequency with which the most unlikely candidates are found to show interest and talent in the exercise of the arts in labor industry or for the sheer enjoyment of accomplishment Manual laborers often show a surprising ability in creative art and sedentary or intellectual workers exhibit considerable manual dexterity at their hobbies

Study Programs Bibliotherapy and Reeducation.—Just as occupational therapy provides a preoccupation with manual accomplishment so the mind of the patient is often stimulated and directed into more proper channels by study programs reeducation and reading These devices are of particular importance in the management of the maladjusted adolescent If the physician has got out of touch with the available reading material he may call upon local members of the teaching profession or the librarian in the neighborhood to assist in arranging a reading list

Religion and Philosophy—Older patients and particularly those who are nearly at the end of their earthly existence often strive for a star to which they may hitch their wagon The safest and best expedient is organized religion If the local clergyman exhibits an interest in problems

Human beings are frequently buffeted by dire and tragic events beyond medical assistance These may be endured with fortitude through the support and understanding of the physician friend The patient ill with pneumonia derives more comfort from the knowledge that his doctor is sleeping in the house or sitting at the bedside than from many of the 'stimulants of the pharmacopeia The physician must not underrate or be heedless of, the therapeutic effect of his presence alone even though he may be embarrassed by his impotence with the more tangible therapeutic modalities Sympathy in the practice of medicine is like the air on the inside of a tire There may be nothing to it but it eases many a jolt and saves many sore spots" (*Neighbors*)

Persuasion and Suggestion —Persuasion and suggestion are powerful psychotherapeutic modalities Most of the 'faith healers employ these agencies with deliberation and cunning

It is morally wrong for the practitioner to exploit the patient who has been given an abiding faith but there is nothing culpable in the use of persuasion and suggestion when these measures are indicated The practitioner may for example urge greater understanding between parent and child or child and parent he may attempt to get a toper on the water wagon he frequently has to urge an errant marital partner to return to his home He attempts to cajole the obese into dieting the chain smoker into the temperate use of tobacco the overconscientious worker or housewife into seeking a vacation or relaxation and the sluggard to assume the responsibilities of everyday life

Persuasion largely uses intellectual forces to oppose the emotional factors involved in the production of conflicts and symptoms Unfortunately the intellect is frequently a puny instrument with which to regulate behavior and feeling The experienced learn not to expect a great deal from appeals to intelligence logic or a sense of decency *Suggestion* is helpful in many anxiety conditions Often the timorous and insecure require a symbol to guide their decisions strengthen them in their purposes and sustain them in hours of evil This symbol may be an intangible vote of confidence from the doctor a placebo or a vitamin pill

Advice —A hazardous but alluring form of psychotherapy is the giving of advice This two edged psychotherapeutic agency appears to be simple and confers a great sense of power on the advisor who usually projects his own whims or attitudes in a given situation The physician who is a total abstainer entertains a different attitude toward alcohol from his colleague who enjoys a cocktail or highball with his evening meal The golfing yachting or gardening enthusiast sees his hobby as a solution for all problems The happily married benedict swears by matrimony but the physician with a nagging wife and ubiquitous mother in law advocates divorce or single blessedness If advice is to be given the physician should attempt an objective attitude shelving for the time being his own likes and dislikes

Advice must be within the patient s potential capacities A day laborer cannot of course take up golf and the city dweller cannot do gardening A housewife cannot take the day off if there is no one at home to prepare the evening meal and make the beds nor can her wage-slave husband take a weekend off or go sailing before it gets dark

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that bridge the narrow gap between medicine and theology he may prove of immeasurable assistance in the management of those who stand in the shadow of death Not only does he comfort the afflicted but his good offices relieve the practitioner of a heavy and exhausting burden It is a wise practice for the physician irrespective of his church affiliation or lack of one to cultivate the local clergymen of all denominations so that he and his patient may count on their cooperation in the resolution of common problems

The unbelievers and those not of formal religious denomination are often comforted by an introduction to the writings of the great philosophers

Remotivation Sublimation and Substitution —Among the well to do patients in any practice there is a vast amount of difficulty that arises as the result of idleness and the pursuit of false gods Few successful men and women and fewer who inherit wealth are able to tolerate good fortune Easy money and leisure invite multiple and recurrent fractures of the Ten Commandments School children and college students on their prolonged holidays tend to get into all manner of difficulties merely for want of something better to do Alcoholism the excessive use of nicotine sexual aberrations and all manner of mischief may be the result of boredom and lack of something to do While the leisure class has more opportunity to cater to such weaknesses it has no monopoly on this commodity Precisely the same conduct disorders and neuroses are observed in the underprivileged and middle classes Whereas the pampered sot ends up in a Turkish bath the impoverished one finds himself in the local cooler or at the business end of his wife s broomstick Who will say which is better and more effectual therapy?

The compleat practitioner often is enabled to accomplish great things by practical remotivation The indolent and indulged housewife may substitute good work in the local philanthropic organizations for her card games and gossip Impressionable youngsters of school or college age sublimate their restless energies when in place of excessive indulgence in athletics or dance contests they are given responsibilities by an ingenious parent or physician

Autopsychotherapy —The greatest and most successful therapist for each patient is the doctor whose face appears in the mirror The accomplishment of any other physician is apt to be transitory and shortlived The patient who can be influenced to exhibit firm self control and will power has surely combated many difficulties that can be influenced in no other way These truths are best illustrated in the treatment of alcoholism excessive smoking and the obesity that results from hyperalimentation In the latter instance all the diet lists in the world are ineffectual in the face of self indulgence Contrariwise the accomplishment of weight reduction is a simple exercise in the management of the patient who can and will adhere to a simple regimen

The appeal to autopsychotherapy serves as a therapeutic test Failure of the method as in alcoholism suggests the presence of a profound neurosis requiring technical assistance from the specialist consultant

Placebos —Many doubts seem to exist in the minds of practitioners relative to the ethics and advisability of employing placebos While such

methods have been attacked by dwellers in ivory towers they serve a beneficial role in the hands of the modern physician We are rather inclined to doubt the sincerity of any practitioner who asserts that he does not employ them consciously or subconsciously This defense of placebos and suggestion is not to be interpreted as a brief for their unjustifiable exploitation in the hands of the unscrupulous within and without the medical profession An untutored and anxious patient must often be given something tangible to take The hurried and harried practitioner often resorts to a placebo when he has not the time to listen to or deal with an anxiety situation

The placebo may be employed for some purpose other than what appears to be its intent The patient may faithfully inhale steam when Tincture of Benzoin is added to the kettle but think poorly of the same therapeutic measure without the odor He may conscientiously soak a limb in a saturated solution of epsom salts when the same procedure would be neglected in tap water A child will gargle with a solution containing aromatic oils but not with saline A patient will return to the physician s office for hypodermic injections but not for further conversations that deal with a difficult situation

The addition of suggestion to the placebo is not without danger When the physician intimates to his patient that the administration of a remedy should or will result in alleviation of the symptoms he gambles with the patient s faith and his own reputation The afflicted sufferer does not forget nor forgive the continuance of symptoms for which relief has been promised He may harbor an antagonism for the physician as an individual and for the practice of medicine in general Those with the greatest capacity for faith have the greatest capacity for cynicism

When it becomes necessary to use a placebo or suggestion the physician hopes that the expedient will prove to be temporary He strives to build a firm relationship with his patient so that he can deal forthrightly honestly and directly in his future therapeutic endeavors

Adjustment of Family and Business Difficulties —At times the emotionally disturbed patient suffers from the shortcomings or afflictions of those with whom he is in contact A husband may be driven to desperation by a neurotic wife who has all manner of compulsions and obsessions so that his life at home is unbearable particularly at the end of an exhausting day of work The woman whose husband has a profound sexual neurosis rendering him impotent may be restless and irritable and sleepless and unstable to the point where she seeks medical assistance She requires relief from symptoms that are referable to her lord and master while he because of the nature of his affliction shuns the physician and denies any difficulties

In the business world the anxiety and symptoms of exhaustion of an employee or partner may be traced to a compulsion neurosis on the part of the head of a business whose driving power is more than his normal integrated associates can endure In academic life pupils often get into serious difficulties as the result of a neurotic tendency of the teacher who strives to compensate for frustrations by beating down the ambitions of young hopefuls In their own lives physicians see these mechanisms at work in hospital politics where the neurosis of one successful chief of

service may cloud and disturb the lives of most if not all of his junior associates

In situations such as these the practitioner is faced with a grave difficulty Under ideal conditions he attacks the problem on two fronts He attempts to desensitize his patient so that he is able to endure that which cannot be cured In a compact community the samaritan physician may also attempt to deal with the difficulty at its source and gratuitously consult with the first cause ' More often than not despite good intentions he leads with his chin and is fortunate indeed if he escapes with minor cuts and bruises

The Use of Drugs —The use of drugs in psychiatry is of minor significance *Sedatives* and *hypnotics* are useful in the treatment of conditions characterized by nervousness restlessness and insomnia Often however this therapeutic endeavor proves a two edged sword since the patient with a neurosis or psychosis is prone to become dependent upon the drug requires increased dosage and develops addiction A more serious complication particularly in the management of epilepsy is the superimposition of *chronic bromide intoxication* with its train of psychotic manifestations often of graver significance than the fundamental disorder

Most of the so called *nerve stimulants* owe their reputation almost entirely to suggestion Manganese phosphates neurophosphates lecithin strychnine caffeine and the like accomplish nothing that is pharmacologically demonstrable and they may be harmful *Amphetamine sulfate (benzedrine)* has proven a real boon however when judiciously used as a cerebral stimulant and *thiamine chloride* is a powerful stimulant to afflicted peripheral nerves

Consultation —For the vast majority of patients the simple devices outlined above suffice for an adequate therapeutic result In a small number of cases a more penetrating and basic form of psychotherapy becomes essential Because of the stigma and opprobrium which still attaches to mental disease the family practitioner is often understandably reluctant to suggest psychiatric help Some patients and many families resent this advice but when the physician has thoroughly exploited his resources to no avail it is his duty to refer the patient to a competent psychiatrist for formal treatment (p 1324) Failure of nontechnical psychiatry exposes the depth and rigidity of the neurosis serves as a therapeutic test and indicates the necessity for formal consultation and treatment

DETERRENTS AND IMPEDIMENTS TO PSYCHOTHERAPY BY THE GENERAL PRACTITIONER

It is unfortunate that psychotherapy by the general practitioner occupies the top of the list in the enumeration of his services for which he reaps ingratitude The patient who is emotionally disturbed is most profligate of the time energies and resources of his doctor and usually manages to upset an entire office hour an evening of anticipated rest and relaxation a holiday or a vacation

It is characteristic of the mental patient that he shifts doctors for no reason other than to manifest his fundamental insecurity to get revenge for what he considers lack of sympathy on the part of the previous listener or out of plain orneriness Add to this the fact that neither the

patient nor his family regard nontechnical psychotherapy as a service of sufficient dignity to warrant the payment of a fee and there results a circumstance in which the practitioner makes a maximum investment of time energy and skill for a minimum fee and a plethora of abuse and ill will

It would be misleading to indicate however that this gloomy prospect is unrelieved Increasingly as the public has become educated the cured neurotics and those who have been helped with their emotional and psychogenic problems become the loyal constituents and supporters of their doctor With the passage of time the practitioner regards with great satisfaction the souls he has saved through a conversation a bit of timely advice or a word or gesture of encouragement and faith given at just the right time The little boy who lied and stole at school becomes distinguished for courage and daring the little girl whose sexual curiosity made her the community plaything blossoms forth as a devoted mother and wife the insecure stripling finds a new world under a microscope in the field of music or science and comes of age the wastrel discovers that fun ain t fun and diverts his excessive energies into purposeful and useful endeavor Behind these minor miracles is often the figure of the doctor who did the right thing and said the right words at the right time often without any regard for the formalities of psychiatry or current concepts of habits and morals

The family physician plays down and even apologizes for his psychotherapeutic endeavor He is intimidated at least in part by the evolution of involved complicated and exhaustive formal psychiatric technics and their mysterious terminologies

Physicians who have their own emotional difficulties are often prevented from adequate psychotherapy by unsurmountable resistances and prejudices Some persist in the concept that emotional sickness is blameworthy They seem unaware of the anguish and waste of an emotional illness and the difficulty of reaching the problems of the sufferer Such physicians are relieved when freed of the onus of taking care of the patient with a psychogenic disturbance If the burden must be assumed the neurotic tendencies of the practitioner often make him unsympathetic and impatient and he projects his own difficulties into the problems presented by the patient thus unwittingly causing considerable damage

The general practitioner also is handicapped in his role of psychotherapist by his intimacy with the patient and the family circle It is difficult to discuss an emotional problem concerning which a patient has a sense of guilt or inadequacy with a companion or neighbor with whom there must be social intercourse Conditions analogous to these lead the psychoanalyst to refuse to treat friends or relatives The patient speaks more freely to a stranger Yet in many instances the practitioner because of personal intimacy with the background and home life of his neighbor patient knows the entire problem without being told and is enabled to cut corners and drive directly to the heart of the difficulty Much of success or failure in dealing with ethico social difficulties depends upon the attitude of the physician If he is known as a regular fellow and foregoes moralizing and attitudinizing the patient finds little difficulty in the discussion of troublesome emotional situations

A practical consideration relative to psychotherapy by the practitioner is the inroad that is made upon time and privacy A single patient with an emotional or situational difficulty may consume a disproportionate amount of time during office hours To give an adequate amount of undivided attention, the busy doctor must often make a special appointment after office hours in the evening or over a holiday thus encroaching upon his own relaxation and diversion

A real mental hazard to the practitioner in informal psychotherapy is the captious and often unjustifiable criticism that sometimes emanates from the formal psychiatrist who later deals with the more seriously afflicted patient Hearing only the version of the newly acquired complainant and not that of the referring physician the specialist is apt to make statements to the patient and his family that are far from tolerant or charitable relative to previous therapeutic devices

Finally, many physicians are discouraged because of their failure to obtain psychotherapeutic results They do not realize that successful treatment of a profound psychiatric disorder demands as much special skill as a major surgical procedure Often they underestimate the patient's wish to be ill' and his tenacity in clinging to neurotic symptoms In many cases forces are at work which require preservation of the illness and the latter is unconsciously exploited in order to derive secondary advantages which would be dissipated by a cure Thus the matriarch whose heart attack occupies the entire attention of her husband and family (whom she has alienated by other defects of character) would indeed be a disgruntled and neglected object were she to lose her symptoms An older child in the household with an infant might be quite desolate but for the attention derived from an attack of vomiting a belly ache or a tantrum

THE PSYCHIATRIST

It would be impossible to refer to the psychiatrist each patient who suffers from a functional nervous condition The anxiety neurosis *mild depression hypochondriasis and situational* and characterological difficulties are the everyday experience of the average practitioner who deals with them to the best of his ability (p 1316) In his routine examination the mental capacities and attitudes are noted The services of the psychiatrist are sought in dealing with the following clinical problems

1 *The patient suspected of having a psychosis* This is done for the protection of the patient the family the community and the physician
2 *Institutionalization of mental patients* The psychiatrist is an expert in problems relating to institutionalization of the patient He possesses intimate knowledge of the best type of institutional care for the individual patient Consultation is mandatory before commitment can be accomplished in most states
3 *Psychometric tests* (p 1325) for the evaluation of mental development where retardation is suspected
4 *Behavior problems among children*

5 *Attempted suicide* (p 1362) Any patient who has attempted sui cide should be seen by a psychiatrist
6 *Drug addiction* including *alcoholism* (p 3851)

Psychometric Tests —Psychometric tests are done for intelligence and personality inventories and for diagnosis

Intelligence Tests—The *Army Intelligence* and the *Terman Binet* tests sample a number of different cerebral functions The patient is presented with a list of test items to measure memory reasoning and imagination He is asked to repeat lists of digits define words point out similarities or differences between objects and solve simple problems The score is compared with the scores of typical and different age groups The patient is assigned a mental age (MA) signifying the age group which his performance resembles

By dividing the mental age (MA) by the chronological age (CA) the intelligent quotient (IQ) is obtained The normal IQ is considered as 90 to 110 and includes 60 per cent of the population An IQ of 80 to 89 is considered low and is typical of 14 per cent of our people whereas 70 to 79 is a border line range typical of 5 per cent Only 1 per cent of the people have an IQ below 70 These are classified as feeble minded A range of 50 to 70 is typical for morons 25 to 50 for imbeciles and 0 to 25 for idiots An IQ of 110 to 119 is high and is attained by only 14 per cent of the people From 120 to 129 is superior and reached by only 5 per cent of the population Anything over 130 is found in the highest 1 per cent of all

Since the tests involve various functions it is possible to subdivide the groups and obtain differential measures of memory imagery comparison reasoning comprehension practical judgment ideational judgment sen sation and perception of form

The *Kuhlmann Anderson* is a pencil and paper test and can be admin istered to a group As in the Terman Binet test there is a battery of sub tests measuring arithmetic reasoning vocabulary etc The scores are translated into mental age (MA) chronological age (CA) and IQ (in telligence quotient)

The *Army Alpha* test resembles the Kuhlmann Anderson The *Army Beta* test is a foreign language test for those who do not speak English the *Sequin method* is non language and is employed in the lower levels of intelligence

Personality Inventories—Of the personality inventories the *Bern reuter* is a paper and pencil test containing 125 items The subject is asked to answer yes or no to such questions as Do you day dream fre quently? Are you slow in making decisions? Do you heckle or ques tion a public speaker? etc

Through a complicated system of differential statistical weighing four different types of behavior are noted The *BIN* is a measure of neurotic tendency Persons scoring high on this tend to be emotionally unstable A second or self sufficient group prefers to be alone and rarely asks for sympathy and encouragement These tend to ignore the advice of others In the introversion extroversion group persons who score high tend to be introverted they are imaginative and live within themselves Those scor

ing low are extroverted rarely worry seldom suffer emotional upsets or substitute day dreaming for action The last is the dominant sufficient group A high score in this category indicates a tendency to dominate others in face to face situations A low score indicates a submissive tendency The various traits are interrelated and are not independent modes of behavior There is a strong tendency for the neurotic and introvert to go together and a lesser tendency for self sufficiency to accompany dominance

The second type of personality inventory is the *Pressey X C test* which is a paper and pencil exercise containing three groups of words Each group has twenty five lists of five words each In the first group the subject is asked to cross out everything he thinks is wrong or blameworthy In the second he crosses out everything about which he has worried has felt nervous or anxious In the third group he crosses out everything he is interested in

On completing the three groups the subject reviews the list and marks that word in each list from which he has obtained the most reaction The test gives two scores The first is an *affectivity or emotionality score* and involves the total number of words crossed out The second an *idiosyncrasy score* is obtained by noting the number of words crossed out in each line that are not the model words for that line

Diagnostic Tests—Of the diagnostic tests the *Rorschach* is the most interesting It attempts to answer the questions Where does the subject look for his meanings? and What meanings does he find? To test these he is shown a series of standard meaningless ink blots Most of them are black but a few have splotches of color The subject looks at these blots one at a time and tells the tester what they resemble or of what they make him think By studying the reported meanings of these figures the expert gains insight into behavior

The test embodies the principle of a free response to an unconventionalized stimulus If the subject s spontaneous interpretation reflects his way of thinking and living a study of these responses reveals much about the organization of an individual personality and his approach to his environment The *total number* of responses gives an indication of the facility of thinking The *quality* of the responses reflects the intelligence and culture level Some interpret the blot as a whole (scored as a *W response*) indicating a tendency to synthesis and organization Others react to some particular detail (*scored as a D*) *showing an emphasis on the particular* and an inability to synthesize The subject may make a great number of *color responses* (*C*) indicating impulse or affective tendencies The blot may be interpreted in terms of movement (*M*) indicating a tendency to inner life and inner living The relation of *C* (color) to *M* (movement) gives the experience balance which indicates a hypomanic personality *If M* overbalances C it is an evidence of inner living typical of the schizoid

Consideration of the interpretive responses permits the expert to draw positive conclusions regarding the type and level of intelligence the quality and intensity of emotional reaction and the degree of personality integration In the hands of the experienced this seemingly simple device is an interesting and valuable procedure

Other Tests—The *Babcock test for mental deterioration* is based upon

the vocabulary ability By comparing this with other mental processes (such as learning comprehension and repetition) the extent of deterioration may be determined since it has been observed that the vocabulary acuity shows few effects of deterioration whereas other elements in intelligence show more An *efficiency index* is obtained depending upon whether the general intelligence is below or above the norm in relation to vocabulary ability A minus index indicates deterioration

The *Jung Free Association test* tends to uncover what is going on in the patient s mind by recording his responses to a list of common words The tester carefully pronounces each word and the subject responds with the first word he thinks of The associative material often furnishes important clues to the individual thought content In addition to the subject content the lag in response is of importance The subject s reaction time may lengthen with emotional difficulty When a stimulus word is accompanied by a prolonged reaction time it may be taken to indicate that the stimulus words possess some emotional significance for the subject

FORMAL PSYCHOTHERAPY

Within and without the medical profession practitioners of psychotherapy vary between the charlatans who practice faith or mental healing and the trained psychiatrist Unlicensed nervous and mental healers are ubiquitous psychotherapy being the most fertile field for the exploitation of patient and family by the unscrupulous the ignorant and the opportunist The practitioner must acquaint himself with the various cults so that he may guide his patients through the morass of mental illness In this capacity he courts ill will to say the least and possible lawsuits for libel at the worst It is futile to expose the infantile concepts of schools of faith healing or to dissuade a lovesick maiden or sex starved spinster from consulting a particularly virile looking Yogi psychologist astrologer fortune teller palmist or crystal gazer

In proportion to the prevalence of nervous and mental disorders the number of trained psychiatrists in the United States is pitifully small Their ministrations are necessarily time consuming arduous and prolonged and more often than not patients resent being referred to them

The technics of psychotherapy are varied and manifold Many of the skilled and successful specialists employ highly individualized entirely eclectic methods and obtain gratifying results more through dint of personality than methodology The formal therapeutic devices of proven value in the treatment of nervous and mental disorders include distributive analysis hypnosis hypnagogic reverie psychoanalysis shock treatment *hyperpyrexia specific therapy and institutionalization* To this armamentarium the surgeon has added the performance of pre frontal lobotomy

Distributive Analysis—Distributive analysis formulated by Adolph Meyer is a relatively simple technic of psychotherapy It consists of persuasion reeducation the detailed discussion of the life history confession and ventilation reassurance and guidance It lays emphasis in contra distinction to psychoanalysis on conscious problems and environmental factors It is particularly useful in the psychotherapy of psychotic patients and its relative simplicity makes it applicable to large numbers of psy

chiatric patients It constitutes a more formal and ordered organization of the methods used by the practitioner

Hypnosis—Treatment by hypnosis is a dangerous undertaking except in the hands of the most reputable and expert It should be avoided by the practitioner

Hypno narco analysis (Hypnagogic Reverie)—The exigency of war has confronted psychiatrists with the necessity of treating large numbers of acute neuroses characterized by amnesia Efforts to penetrate the amnesia are made by hypnosis psychoanalysis and the hypnagogic reverie The last is combined technic which incorporates the use of sedative drugs in order to render the subject accessible to hypnotism The exploration of the unconscious is then attempted for purposes of diagnosis and therapy

The hypnagogic reverie may be induced by simple suggestion the use of sedative drugs or the rhythmic auditory stimulus of a metronome The recommended drugs include sodium amytal 0 2 gm orally every three or four hours sodium amytal 0.5 gm in distilled water administered intravenously, pentobarbital sodium (nembutal) injected intravenously at a rate of 1 cc per minute using 2½ per cent solution sodium evipal 0.3 gm orally, sodium pentothal given intravenously as in anesthesia (p 3023) and a combination of scopolamine hydrobromide 0 5 mg with morphine sulfate 15 mg by subcutaneous injection

During the hypnagogic reverie amnesic material is recovered and fully fused with its appropriate emotional content and with normal waking consciousness, the findings are reworked with the patient as he emerges from the hypnoid stage

The hypnagogic reverie involves technics that are too complicated for the general practitioner who need merely be aware of the existence of the method so that he may refer suitable material to the experienced psychiatrist

Psychoanalysis—Psychoanalysis has been the chief impetus in the growing interest in modern psychotherapy So greatly has this important innovation influenced the literature and thinking of our time that it is important to define the procedure and indicate the qualifications that are necessary for its practice

In general the reputable psychoanalyst is a graduate physician who is trained in general psychiatry He has supplemented his medical education with a training analysis by an older and accredited psychoanalyst and has himself analyzed at least a few patients under guidance and tutelage before embarking on his separate career Trained analysts are found only in the larger cities and in limited numbers so that the technic is available to only a minute proportion of the population Self-constituted 'analysts who do not have proper qualifications are to be avoided since inexpert treatment may produce grievous results

It is customary for the patient to recline on a couch so situated that the analyst seated in his chair is beyond the visual field The patient then allows his thoughts to ramble without a systematic presentation of his ideas In this free and aimless fashion symptoms are described the life history and current situational problems emerge and fantasies emotions and dreams are set forth In this way many disturbing ideas and feelings come to verbal expression at the same time the undirected

stream of thought brings to light many deeper connotations and opens the way to an exploration of underlying hidden mechanisms forgotten experiences and buried and troublesome strivings

During these sessions the analyst attempts to keep himself and his personality as much in the background as possible In this way the analysis simulates the conditons of the religious confessional in which the priest unseen by the penitent maintains anonymity The analytic patient displaces onto his analyst all of the complicated feelings which are entertained relative to those who are important in his real life This shift of feelings to the analyst is called *transference* and it is compounded of love hate fear and all other human reactions expressed in many disguised forms It is an essential part of the work of the analyst to investigate these feelings so that the patient may acquire insight into the underlying nature of his human relationship

As the analysis proceeds deeper and deeper layers of the unconscious are covered Infantile phantasies are revealed the meaning of symptoms becomes clear incongruous conduct becomes apparent and the emotional tones *attached to the various symptoms are abreacted and dissipated* Generally the mere bringing to the surface of emotionally charged associations serves to free the symptoms the patient no longer clings to them their meaning is understood they cease to have an emotional hold and in the successful analysis they vanish

As transference to the analyst constitutes a *substitution neurosis* the final object is to free the patient from the attachment take away the props on which he is leaning and educate him to face the world of reality on his own feet At first the analyst represents the world of reality onto whom the infantile fantasies are attached By degrees the patient learns to shift his interests away from the analyst to the world of objective reality sublimates his energies and learns to face situations in an adult manner A successful analysis then consists of a *reeducation reintegration* and *reorientation* of the whole personality The energy is freed and directed into artistic intellectual and moral pursuits thus creating a new goal the patient gives up his neurosis gains a sense of reality from the upbuilding of the real ego and is prepared to meet difficult situations in life in an objective manner

In our limited experience the best results are obtained in the simpler neuroses and conversion mechanisms the profound neuroses and psychoses are little influenced *The technic of psychoanalysis requires attendance for* approximately an hour daily for at least eighteen months i.e s
400 sessions The fee for each treatment may vary from 25
an hour At $5 00 an hour 400 sessions cost $2000

Shock Treatment.—The recent innovation or re
in the management of the psychoses constitutes
peutic advances of the present era
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injection of medullary convulsants such
specially devised *electrical equipment* The
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In our limited experience the best results are obtained in the simpler neuroses and conversion mechanisms the profound neuroses and psychoses are little influenced The technic of psychoanalysis requires attendance for approximately an hour daily for at least eighteen months *i e* perhaps 400 sessions The fee for each treatment may vary from 25 cents to $25 an hour At $5 00 an hour 400 sessions cost $2000

Shock Treatment—The recent innovation or rebirth of shock treatment in the management of the psychoses constitutes one of the greatest therapeutic advances of the present era Convulsions may be induced by the production of a hypoglycemia through overdosage with *insulin* by the injection of medullary convulsants such as *camphor* and *metrazol* and by specially devised *electrical equipment* The danger of fracture due to the violence of the convulsions may be lessened by a prior intramuscular injection of *intocostrin* a curare preparation in a dose of 1 cc to 20 pounds of body weight The total dose should not exceed 4 cc See p 1330

The use of these modalities is specialist province under institutional conditions Splendid results have been obtained in *simple depressions* and *manic depressive states* less spectacular remissions are seen in *schizophrenia*

Insulin Shock —For the induction of hypoglycemic shock the patient is institutionalized in a hospital where the staff has become familiar with the technic After a fast of twelve hours the patient is given an initial probatory dose of 15 units of soluble insulin The unitage is increased daily by 5 or 10 units until the signs of hypoglycemic coma begin to appear If coma does not develop three or four hours after the injection the patient is given a solution of sugar to drink if necessary dextrose is injected intravenously

When the induced hypoglycemia reaches the level at which the patient loses consciousness on or about the third hour the unitage is kept stationary and repeated six times weekly for two or three months Each hypoglycemic episode is terminated by giving sugar by nasal tube or intravenous injection A satisfactory response is characterized by loss of consciousness in the third hour and deep coma in the fourth or fifth hour The patient should not be permitted to stay in the deep coma for more than 45 minutes On awakening a substantial meal of carbohydrate is ingested

Metrazol Shock —Metrazol shock is produced by an intravenous injection of 3 to 5 cc of a 10 per cent solution Within 5 to 15 seconds a convulsion is inaugurated The motor response may be so violent as to cause fracture of the jaw or a vertebra In consequence the metrazol convulsion may be eased by the prior use of intocostrin a curare derivative which does not interfere with the convulsion but which softens the muscular response See p 000

If the initial dose of 3 to 5 cc of metrazol is ineffectual the amount is increased each time by 0 5 to 1 cc until the convulsion is produced Metrazol treatments may be given twice weekly it is wise policy to limit the number of convulsions to a total of eight

Electric Convulsive Therapy—Several machines have been developed to apply alternating current to the fronto temporal regions for the production of convulsions Following the passage of the current the patient may have a minor or major seizure with loss of consciousness for a few minutes Intocostrin may be used (p 1329) to prevent injury due to the violence of the seizure or preferably the treatment may be given after intravenous anesthesia with sodium amytal 0.5 gm (7½ grains) (in 10 cc of diluent)

Following the electrically induced convulsion the patient is restored to consciousness in 30 to 35 seconds and usually exhibits no after effects other than some amnesia Electric convulsions may be given twice weekly for a total of eight to twelve treatments

Hyperpyrexia —The use of induced fever has proven a therapeutic boon particularly in the treatment of *general paresis* of syphilitic origin The state of hyperpyrexia is induced through artificial infection with tertian or quartan *malaria* through intravenously injected *foreign protein* (typhoid vaccine) or by mechanical devices such as the *hypertherm*

These therapeutic measures are specialist province and not without danger even under optimum conditions

Specific Treatment—The organic psychoses of specific origin are treated by methods directed at the fundamental etiologic mechanism Thus syphilis of the nervous system is attacked by anti syphilitic drugs and the psychoses that result from avitaminosis require the feeding or injection of the necessary metabolic agencies (p 1375)

Institutionalization—In general the practitioner recommends institutionalization for those whose illnesses preclude their useful function in the community or who are destructive of their own their family and community interests

The practitioner must inform himself concerning the *medicolegal technicalities* that arise in the institutionalization of patients These formalities differ in the various states Local regulations may be obtained from police or legal authorites and a few well directed inquiries may save the practitioner a great deal of criticism trouble and even litigation

Neurological and psychiatric hospitals vary between the splendid reliable humane and scientific establishments (altogether too few in number) and the neurological boarding hou es run by charlatans and incompetent or unethical practitioners

It is often a matter of considerable difficulty to persuade relatives to commit the psychotic The resistance is usually greatest in dealing with mental disorders that involve children or elderly parents Often many lives are affected and even ruined by the insistence of the family that the afflicted must not be put away Tremendous economic sacrifices are made to maintain the patient in an incompetent private sanitarium when far better provision could be afforded in a municipal state or federal institution supported by the taxpayer

Pre frontal Lobotomy—The operation of pre frontal lobotomy has been devised in an effort to deal with the psychoses Holes are drilled in the skull and the thalamo frontal radiation is severed with a leukotome passed through a cannula In expert hands the procedure is not dangerous and an occasional spectacular result has been obtained in deteriorated psychotics who have failed to respond to all other types of therapy The most favorable results are reported in obsessive tension states hypochondriasis and agitated depressions

CHAPTER 63

PSYCHIATRY MENTAL DEFICIENCY

IDIOCY imbecility and moronity are the three levels of feeblemindedness See *Psychometric Tests* (p 1325)

Idiocy—An idiot is a person with a mental age of two years or less and an intelligence quotient between 0 and 30 Idiots are unable to care for themselves even to the extent of dealing with common physical dangers In a sense they are more like animals than humans They can utter but a few monosyllables and are frequently unable to walk

Imbecility —The imbecile has a mental range of from three to seven years and an intelligence quotient that varies between 30 and 50 Imbeciles are incapable of managing for themselves on anything beyond the most primitive level They can learn simple toilet habits and are capable of feeding themselves but beyond these functions they are quite helpless

Moronity—The moron has a mental age of from seven to twelve years and an intelligence quotient that varies between 50 and 70 Moronity does not constitute so striking a deviation as idiocy and imbecility Moronity may escape attention until schooling begins and the backwardness is discovered through the failure of the subject to learn and progress

Management—The management of the feebleminded child poses a difficult problem for the practitioner Under no circumstances should he attempt to render any judgment without the assistance of the expert A complete neurologic and psychiatric examination is mandatory *Psychometric tests* (p 1325) are performed the disposition of the child depends upon the findings translated into terms of emotional social and economic factors in the household situation

There is obviously no specific form of therapy that can be employed It becomes necessary to confront parents with a realistic view of the situation and assist them in making an adjustment for the unfortunate child and themselves The practitioner attempts the difficult tasks of protecting the child getting the most out of the handicapped life and preventing bewildered and well intentioned parents from ruining several lives in a futile effort to save one The continued presence of an idiot or imbecile in a home may result in financial sacrifices on the part of the parents the exertion of a deleterious influence on other children growing up in the household and a complete submersion of all other activities to the protection of the hopelessly handicapped child

Institutionalization —There is little doubt that institutionalization fur nishes the best outlook for idiots and imbeciles This advice always meets with the most violent resistance particularly on the part of the mother

DIFFERENTIAL DIAGNOSIS OF

Feeblemindedness

The suspicion of feeble mindedness warrants specialist consultation with the trained neuro psychiatrist. On rare occasions therapy may be initiated with some small hope of success but in all instances the practitioner owes it to him elf to be protected against criticism in the face of these overwhelming tragedies

CAUSE	DIAGNOSTIC FEATURES
Cerebral Birth Trauma	Spa ticity palsy and athetoses (p 2771)
Erythroblastosis Foetalis	Icterus gravis Hemolytic anemia due to ab normalities of the Rh factor
Mongolian Idiocy	Characteristic obliquity of eyes (p 2774)
Microcephalus	Early closure of fontanelles and small cir cumference of head (p 2774)
Cretinism	Thickening of skin protrusion of tongue and umbilical hernia Responds to thyroid extract (p 1191)
Hydrocephalus	Pumpkin Head Refer to neurosurgeon for possible palliative procedure (p 2774)
Cachexia	In chronic disease particularly tuberculosis endocrinopathies and malignancie
Congenital Syphilis	Positive serologic findings in maternal um bilical and infant s blood (p 2787)
Anencephaly	Absence of brain
Porencephaly	Defects of cerebral hemispheres (p 1407)
Cerebral Agenesis	With palsies ataxia and cranial nerve invol e ment (p 1408)
Amaurotic Family Idiocy	With characteristic cherry red spot in fundu (p 1412)
Niemann Pick Syndrome	Anemia leukocytosis splenomegaly and hepatomegaly (p 1134)
Hand Christian Schuller Syndrome	Diabetes insipidus exophthalmos and defects of bone (p 1137)
Laurence Moon B edl Syndrome	Obesity hypogonadism retinitis p gmentosa and polydactyli m (p 1166)
Tuberous Sclerosis	Associated with cutaneous pigmentation ad enoma sebaceum and fibromas (p 1133)
Huntington s Chorea	Familial disturbance associated with motor disturbances and mental deterioration (p 1417)
Progressive Lenticular Degeneration	Athetosis spasmodic laughter and hob-nail cirrhosis of liver (p 1418)
Aplasia Axialis Extracorticalis Congenita	With nystagmus headache and speech de fects (p 1418)

In some instances her revulsion is justified since many public institutions for the care of the feebleminded are overcrowded understaffed and thor oughl unsatisfactory The better private schools are exceedingly expen

sive and quite beyond the budgetary possibilities of all save those in the upper income brackets

One of the principal uses of the psychiatrist is to obtain his broader knowledge of the local institutions conducted by private enterprise and those which operate under the auspices of the state It may here be stated that it does not necessarily follow that the more expensive excel those whose costs are more moderate Often the retreats supported by the tax payer enjoy facilities that are far superior to those under private management

At times it is necessary for the physician to assist the parents in their attempts to keep the child at home He understands that there are imponderable emotional aspects feelings of guilt and other unconscious attitudes that lead parents determinedly to oppose institutionalization of the deficient child If the necessary sacrifices are not excessive it may be wiser to permit the child to remain at home at least temporarily If an extremely modest goal is set for the mentally deficient child the afflicted youngster may function in a socially acceptable manner and may master simple tasks More ambitious goals lead to inevitable disappointment Drastic treatments by shock fever or lobotomy have no promise for benefit

The administration of 8 to 10 gm (120 to 150 grains) of glutamic acid appears temporarily to raise the intellectual levels of non epileptic mental defects

CHAPTER 64

PSYCHIATRY THE NEUROSES

- Etiology of the Neuroses
- Psychoanalytic Concept of the Origin and Pathogenesis of the Neuroses
  - The Unconscious and the Libido
  - Depth Psychology (Ego Id and Superego)
  - Ego and Death Instincts
  - Pleasure pain and Reality Principles
  - Repression and Resistance
  - Conflict
  - Results of Conflict
  - Determination of the Nature of the Reaction
  - Precipitating Factors
  - The Complex
  - The Reactions to Conflict and the Complex
  - Terminology of Neuroses
- Symptoms of Neuroses
  - Anxiety
  - Phobias
  - Neurasthenia
  - Hypochondria
  - Conversion
  - Fixations and Regressions
  - Compulsion
  - Perversions
  - Displacement Projection and Rationalization
  - Dreams and Slips of the Tongue
- Resolution of the Neurosis
  - The Latent Neurosis
  - The Useful Neurosis
  - The Clinical Neurosis
- Clinical Varieties of Neuroses
  - The Anxiety Neurosis
  - Neurasthenia
  - Hypochondriasis
  - Conversion or Transference Neuroses (Hysteria)
  - Traumatic Neuroses
  - Compensation Neuroses
  - Neuroses of Regression and Perversion
  - Mixed Neuroses
- Disorders of Total Personality
  - Personality Behavior and Conduct Disorders

The neuroses have been previously defined as psychological disturbances which arise as reactions to stress they are not accompanied by any serious alteration in evaluation of reality and are partial reactions in that the personality is otherwise well integrated and socially organized

The neuroses are of such common frequency that many practitioners believe that neurotic behavior is ubiquitous Neurotic symptoms usually enter the realm of clinical medicine only when the patient is sufficiently disturbed to seek consultation with his physician However it is the re

sponsibility of the physician to recognize the existence of a neurosis which the patient may attempt to conceal through ignorance or cowardice Acuity of insight and tact on the part of the family practitioner are vital forces in the earlier recognition and treatment of the neurosis years before the patient may recognize the need for assistance Preventative psychotherapy is of as great value and significance as prophylactic immunizations in the infections

ETIOLOGY OF THE NEUROSES

Until the time of Freud the etiology of the neuroses was obscure and investigations failed to lead to any satisfactory therapeutic approach to the management of the afflicted It was generally believed that *hereditary* and *constitutional factors* were of considerable importance since familial examples were the rule Organicists pursued the possibility that the neurosis resulted from subtle and undemonstrable molecular and pathologic changes in the nervous system possibly resulting from prenatal influences syphilis in the third and fourth generation congenital afflictions birth trauma intra uterine infection abortive infections involving the meninges or brain tissue proper and sequels of the exanthemas or vascular abnormalities

Zealous *reformers* attributed the neuroses to excessive or long standing indulgence in alcohol to masturbation or to sexual excesses or sexual abstinence *Physiologists* claimed the affliction resulted from disorders in nerve impulse production and the *endocrinologists* believed that the psychiatric disorder rested upon a dislocation in the interplay of the glands of internal secretion The *gynecologists* clung to etymology and since hysteria is derived from hystera (womb) they corrected uterine malpositions and removed the womb often with a temporary and illusory psychotherapeutic result but with a later profound rage and depression Advocates of the theory of *auto intoxication* sounded their drums particularly in the elucidation of the depressed states and psychasthenia The enthusiasm for *focal infection* led to a wave of tonsillectomies appendectomies cholecystectomies tooth extractions and wholesale removals of the large intestine—all to no good or permanent purpose Students of the *involuntary nervous system* attempted to correlate the vegetative neuroses and psychiatric disturbances and *while it is true that these* coexist it is more likely that both arise from a common stem than that either one primarily causes the other

It remained for Freud to lift the curtain of obscurity and suggest a working hypothesis for the origin of the neuroses His theories and investigations have a rational appeal and a therapeutic approach that hold some promise for benefit to the patient Stated in broadest terms it is Freud s belief that there is a purposive nature to the neurosis the symptoms are not meaningless and psychological factors of repression conflict and persistent infantile behavior patterns figure prominently in the etiology The manifestations serve as a defense against the gratification of unconscious repressed guilt laden desires They represent disguised gratifications of repressed strivings which cannot be permitted conscious expression

The psychoanalytic therapeutic endeavor depends upon the *replace*

ment of conscious deeds for unconscious mental acts The Freudian theory of pathogenesis and treatment are further enlarged in succeeding paragraphs

PSYCHOANALYTIC CONCEPT OF THE ORIGIN AND PATHOGENESIS OF THE NEUROSES

There is universal agreement that the faculties of thought and ideation reside in the cerebrum Prior to the time of Freud the processes of voluntary cerebration were recognized and abnormalities were catalogued without recognition of a possible relationship between sanity and insanity The writings of Freud have emphasized the levels of consciousness the conscious is that portion of our reasonable being which deals with objective realities the preconscious a subliminal state through which unconsciously motivated ideas must pass in order to reach consciousness

The Unconscious and the Libido—The unconscious is a *dynamic force* in which are stored the experiences of the lifetime of the individual They are often charged with emotion and exist not in a quiescent state but in a flux much as the contents of a boiling cauldron or a smouldering volcano All instinctual energies converge to form the *libido* The sexual and aggressive components of the libido are most important since they are most apt to produce conflict with society conscious conscience or fear

The great contribution of Freud has been his assumption of subconscious or unconscious mental processes and his insistence that these are no mere passive devices but rather dynamic influences in the determination of personality dream life conduct behavior and the nervous and mental disorders of clinical practice The study of the science of unconscious mental processes has been termed by Freud *depth psychology*

Depth Psychology (Ego Id and Superego)—Freud regards the mental apparatus as a composite instrument It is made up of a *superficial conscious stratum* concerned with the perception of the external world and a *deeper unconscious* the reservoir of instinctive impulses Conscious and unconscious are possessed of three major components to which Freud has applied the terms Superego Ego and Id The Superego is the conscience processes the Id represents the instinctual energy reservoir and the Ego is that aspect of personality which relates the individual to the external world by perceptual and affector mechanisms Mental processes are derived from the interplay of forces which assist or inhibit one another combine or enter into compromises one with the other

This Freudian concept of the dual or manifold aspects of the human mind is acceptable to thinking physicians Indeed the hypothesis is neither new nor unique so far as Freud is concerned since the concepts of man wrestling with his soul and the portrayal of sacred and profane love have been current in the literature and art of the world since history has been recorded

Ego and Death Instincts—The dynamic depth psychology of Freud regards the unconscious as the reservoir of instinctive impulses It is assumed that the latter include ego and death instincts

The *ego instincts* are directed toward self preservation hence their concern is with the procreative functions Freud has given the name

libido to the forces of eros It is in the elucidation of this "ego instinct" that Freud has suffered most from his critics and overzealous devotees On even the most superficial inspection it is apparent that the Freudian libido represents a great deal more than mere sexual intercourse In its broader significance and in the sense that Freud originally intended the libido is concerned with all factors which integrate for racial self preservation Whereas copulation between male and female constitutes a necessary link in the chain of events there is no disposition in the Freudian psychology to state that the only concern of the libido is relative to sexual union and of itself

Freud also recognizes in the human mind an instinct for destruction which tends toward the dissolution of what is living This *death instinct* not accepted by all psychoanalysts is an outstanding characteristic of many individuals and of certain national traits of the Nazis and the Japanese

It was Freud s belief that instincts had aims or ways of gratification and objects individuals or things towards which the instinct was directed as a goal

Pleasure pain and Reality Principles —It is the psychoanalytic concept that instincts have a definite charge ' It is the function of the mental apparatus to hinder any damming up of energies and to keep as low as possible the total amount of excitation to which it is subject The course of the mental process is automatically regulated by the *pleasure-pain principle* Pain is related to an increase of excitation and pleasure to a decrease In the course of its development the original pleasure principle undergoes a modification with reference to the external world It gives place to the *reality principle* whereby the mental apparatus learns to postpone the pleasure of satisfaction and to tolerate temporarily feelings of pain

Repression and Resistance —In the world of reality the mind is required to exercise the function of censorship over the fundamental instincts It is necessary to exclude from consciousness and from any influence upon conduct those tendencies which displease the conscious mental processes The exclusion of these tendencies constitutes a *repression* The repressed instinctual impulses are not made powerless but they succeed in making their influences felt by circuitous paths and the indirect or substitutive gratification of repressed impulses These devices constitute the symptoms of the neurosis and when the physician attempts to bring these repressions into the patient s consciousness he provokes a *resistance*

Conflict —Since there exists a dynamic unconscious which constitutes the reservoir of instinctive impulses it is inevitable that these should come in conflict with objective reality whose concerns are the artificial barriers that are set up in the external world The infant cannot urinate whenever its bladder is full it cannot defecate merely because the rectal ampulla is stimulated but must wait for an appropriate time and an appropriate place Its instinct is being opposed by conformity to manmade standards The subconscious is at grips with the conscious and the id is being dominated by the superego These divergencies give rise to *conflict* The individual personality his ego-structure with its strength and weaknesses and its compulsive patterns of behavior is hammered out by

the powerful interactions between instinctual forces external reality and the conscious and unconscious conscience of the super ego

Results of Conflict—*The results of conflict manifest themselves in normal* and abnormal human behavior The individual exhibits love hate wishes desires compulsions fears anxiety perversions fixations transferences *displacement* identification rationalization sublimation dreams and conversion Each and all of these are *compatible with the norm* provided that the life love and occupation of the individual are not seriously affected When they involve only a portion of the personality without a change in the reality situation when the patient retains an insight into his affliction although the symptoms begin to interfere with the enjoyment of life or its accomplishments the condition is to be regarded as a *neurosis*

When there is total involvement of personality a quantitative change in reality a regression to primitive levels of behavior with lack of insight and when the direct verbal expressions are the language of the unconscious with delusions hallucinations and disorders in the form and flow of thought the patient is then regarded as being *psychotic* These definitions and differentiations are considered in more detail in succeeding paragraphs

Determination of the Nature of the Reaction—One of the unsolved problems of psychiatry is the elucidation of the factors which are responsible for *individual and normal behavior* Why does *one* patient develop an anxiety another hypertension the third a gastric neurosis and still another a psychosis? This same problem is inherent in the mechanisms that produce *the reaction picture* of autonomic imbalance Present information holds no answer to the problem Many of the psychoanalytically trained practitioners regard the nature of the disturbance as the predominating factor Thus it is thought that the type of conflict determines whether the patient is to develop a gastric neurosis or a retentive or expulsive type of intestinal neurosis The theories which have been put forth relative to this point are far from conclusive though no better explanation is today available

Precipitating Factors—It is quite possible for the neurosis to lie dormant Conflict is present but like a quiescent volcano is apparently in a state *of inactivity though closer scrutiny reveals the turbulent undercurrent*

The *dormant neurosis* is often precipitated by nonspecific exciting causes and then unfortunately the clinical diagnosis intimates that the secondary factor was actually primary Thus the literature speaks of the "traumatic neuroses war neuroses compensation neuroses menopausal and pregnancy neuroses and adolescent neuroses These terms are misleading since they stress the less important mechanisms and cloud the significant disturbances

The Complex—The result of conflict is the development by the patient of a complex which Freud defines as an unconscious group of ideas charged *with various emotions* These complexes indirectly influence normal behavior and often produce inexplicable symptoms Two of the more frequently encountered complexes are described by the psychoanalysts as the castration and Oedipus syndromes

Castration and Oedipus Complexes—The *castration complex* represents an infantile phantasy concerning the loss of or threat to the phallus The

Oedipus complex embodies an identification of the child with the parent of the same sex and a hostile intent against that parent with the objective of displacing him or her in the affection of the parent of the opposite sex

The Reactions to Conflict and the Complex —The reactions of the individual to the complex that arises from conflict are varied The conscious ego which acts as a ' censor' to the subconscious id " may successfully repress the baser impulses The mechanism of repression however is no mere erasure since the complex is held in the dynamic unconscious under pressure It becomes charged with emotion and exists in a flux not in a quiescent state

Terminology of Neuroses —It was Freud s belief that the most intense repressions fall upon the sexual and aggression instincts The repressed libido responded clinically in a variety of ways including the development of neuroses These were capable of subdivision into those which constituted *simple repressions* those characterized by the mechanisms of *conversion* or *transference* and the *regressions* and *perversions*

The principal value of the Freud classification is the grouping of the neuroses on the basis of dynamic mechanisms A more useful arrangement is one which merely groups the more frequent symptom complexes and recognizes (*1*) Neuroses in which anxiety predominates (*2*) those in which there are predominately conversion symptoms (*3*) neuroses characterized by obsessional and compulsive phenomena (*4*) the addictions and perversions which may occur in association with any of the previous varieties

The Simple Neuroses (Actual Neuroses) —Before embarking further into the discussions of the neuroses it becomes necessary to clarify terminology The present usage is to include in the simple neuroses the conditions that are variously described as the *anxiety neurosis neurasthenia* and *hypochondria* They correspond to what Freud calls the actual neuroses

Conversion or Transference Neuroses (Psychoneuroses) —The conversion or transference types of neurosis are called the psychoneuroses by Freud and are subdivided into *conversion* and *anxiety hysterias Occupational* and *war* neuroses are included in the conversion category It is the present purpose to avoid the use of the term hysteria since it has confusing and disturbing connotations It is resented by the laity who regard it as synonymous with malingering exaggeration and thoroughly culpable conduct disorders

Regressive Neuroses and Perversions —The regressive and perversion neuroses constitute the most serious types Included in this group are the *compulsion neuroses* the *obsessional neuroses* and the *perversions*

SYMPTOMS OF NEUROSIS

The common symptoms of neurosis include anxieties phobias neurasthenia hypochondriasis compulsion obsessions conversions and perversions

Anxiety —Anxiety is the most common neurotic symptom It occurs in the anxiety neurosis in anxiety hysteria in the phobias hypochondriasis melancholias compulsions and obsessions The anxiety may be referred to death disaster heart failure suffocation insanity cancer apoplexy coro-

nary thrombosis continued virginity or imminent rape The symptoms occur by day or night An annoying characteristic is the *pavor nocturnus* or horror which may or may not be associated with the nightmare

Anxiety symptoms are most common in the young and occur more frequently in females As objective accompaniments and results of anxiety there appear tachycardia elevation of blood pressure vasodermal phenomena such as blushing or blanching perspiration urination diarrhea vomiting and cardiac irregularities These phenomena in the acute phases give rise to serious diagnostic problems Accurate interpretation requires careful and repeated observation over a period of time

Phobias —The phobias are hysterical fears with a definite ideational content They constitute according to the Freudians an unconscious wish or emotion that has become dissociated from a more vivid desire The virgin who has a fear of men (androphobia) is probably highly desirous of male companionship and anticipates sexual gratification Other instances of phobia seem less clearly defined and many have been labeled by the descriptive school of neurologists (p 1336)

Neurasthenia —Neurasthenia constitutes an abnormal fatigue and resultant exaggerated irritability It occurs most often in the middle aged but it may be experienced by the young Patients complain of tiring weakness fatigability inability to concentrate memory defects pressure on the head pain in the back dizziness asthenopia orthostatic tachycardia and excessive sensitivity to light noise odors or taste The neurasthenic symptoms are often associated with a subnormal temperature low blood pressure low blood sugar tachycardia and more often than is generally suspected a low basal metabolic rate

Often the neurasthenic gives a history of increased sexual desire with excessive sexual activity and inadequate sexual gratification As a result the complaints include chronic masturbation nocturnal emissions spermatorrhea futile erections premature ejaculations and impotence The last occurs at the time of greatest sexual excitation and the optimal opportunity for gratification Of all the neuroses neurasthenia is most likely to progress to schizophrenia and its recognition demands psychiatric consultation

Hypochondria —Hypochondria indicates a morbid preoccupation with organ integrity When it becomes delusional it is an integral part of the psychotic reaction most frequently however it is a generalized frame of mind an incessant worry and half conviction that something is wrong Most often the attention is focused on the erotic zones such as the mouth anus and genitalia Hypochondriasis may be observed in males but is more common in females at the time of the menopause

Conversion —The conversion symptoms constitute the greatest problem in the neuroses and represent the various manifestations of hysteria The variety of the conversions is as manifold as that of the human countenance and the finger prints No two individuals seemingly manifest the same conversion symptoms yet each complex is unique for that particular individual

The repertoire of conversions include sensory motor autonomic visceral psychic and emotional manifestations

Pain —Pain is a frequent conversion symptom and one which gives rise

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or emotional disturbance The psychiatrist dared not trespass upon the field being warned off by the visible ulceration of the mucous membrane *Meantime the gastro-enterologist intent upon healing of the ulcer became* focused upon gastric acidity gastric intubation and lavages with only lip service to the psychogenic disturbance

Developments in the field of *psychosomatic medicine* emphasize the concept of the unity of body and psyche and of their constant interrelationship It is truism old in medicine but recently reemphasized that illness involves the indicated organ and the entire personality In addition to specific emotional factors implicated etiologically in organic illness the reaction of the individual to his illness requires understanding The patient s neurosis may complicate or contribute to the etiology of his disease and on the other hand the organic disease may be exploited neurotically

Essentially the functional theory of organic disorders is the recognition that diseases are caused by internal as well as external factors Franz Alexander* a pioneer worker in the field of psychosomatic relationships asserts Many chronic disturbances are not caused by external mechanical chemical factors or by the micro organisms but by the continuous functional stress arising during the everyday life of the organism and its struggle for existence Fears if repressed result in permanent chronic emotional tensions which disturb the functions of the vegetative organs Many emotions due to the complication of our social life cannot be freely expressed or relieved through voluntary activities but remain repressed and then are diverted into wrong channels instead of being expressed in voluntary innervations without influencing internal vegetative functions such as digestion respiration or circulation There is much evidence to show that just as pathological microorganisms are specific and have a certain affinity to certain organs so also emotional conflicts are different and are liable in accordance with these differences to involve different internal organs inhibited rage seems to have a specific relationship to the cardiovascular system dependent help seeking tendencies seem to have a specific relationship to the functions of nutrition again different and specific conflict betwen sexual wishes and dependent tendencies seems to have a specific influence on respiratory disturbances The increasing knowledge of the relations of emotions to normal and disturbed body functions requires that for the modern physician emotional conflict should become just as real and tangible issues as visible microorganisms

With the more recent popularization of psychogenic factors in clinical medicine an attempt has been made to unite the efforts of the organicist treating the end organ manifestations and the psychiatrist interested in the emotional factor This wedding has been legitimatized under the title of Psychosomatic Medicine

The enthusiasts for psychosomatic medicine must not be permitted to go to the extreme of isolating their specialty The present delineation holds that the psychosomatic manifestations are essentially conversions and hence not different in their origin implications management and t eatment than the neuroses without objective evidences of somatization Psychosomatic implications are considered in the present text in the presentations of the following disturbances

* Psychological Aspects of Medicine Psychosom Med 1 7-18 (Jan) 1939

to great difficulty in diagnosis (p 1474) The more frequently observed painful hysterical afflictions are the *clavus headache* often characterized by a boring sensation in the skull, *coccygodynia* discussed more fully in the section on back pain (p 3072) and *mastodynia* and *glossodynia* which often have erogenous implications

Paresthesia —The conversion paresthesias are characterized by absence of objective sensory findings bilateral and symmetrical distribution and failure of the complaint to correspond to the anatomic distribution of the nerves Thus there are encountered *pruritus* particularly in erogenous zones (vulva vagina rectum) *acroparesthesias*, *hemianesthesias*, sock and glove' *anesthesia* and *hemianalgesias* In the special senses patients note *anosmia amaurosis photophobia myopia micropsia macropsia* and *mutism*

Abnormalities of Striated Muscle —The conversion abnormalities of muscle take the forms of *spasm or paralyses* There occur peripheral *tics* and *tremors contractures* isolated *muscle spasms* and *astasia abasia Peripheral paralyses* occur without changes in the reflexes They may be in the nature of *monoplegia hemiplegia* or *paraplegia* In the realm of the special senses there are observed *blepharospasm pseudoptosis glossolabial paralyses stammering stuttering* and *aphonia*

Autonomic Manifestations — Autonomic imbalance frequently accompanies other of the conversion symptoms *Vasodermal disturbances* are observed as blushing flushing blanching suffusion and moisture of the extremities or the more disturbing manifestation of the cold clammy hands and feet The *cutaneous phenomena* include dermagraphia urticaria angioneurotic edema and the maddening and perplexing varieties of pruritus involving all skin surfaces but more often the anus vulva vagina or rectum

The vegetative disturbance may be reflected in the internal viscera There are thus produced the *gastric and intestinal neuroses* globus hystericus aerophagia cardiospasm anorexia nervosa bulimia pica nervous vomiting borborygmi diarrhea (mucous colitis) and obstinate constipation *Genito urinary manifestations* include vaginismus enuresis perineal spasms, pollakiuria (frequent voiding) urinary retention and pseudocyesis (false pregnancy)

The *cardiovascular neuroses* include instability of the pulse rate or rhythm hypertension hypotension lability of the blood pressure tachycardia cardiac irregularities and peripheral vascular spasms The respiratory symptoms may be tachypnea sighing gasping and the familiar inability to catch the breath The existence of hysterical fever' is possible aside from malingering

Visceral or Psychosomatic Manifestations —Psychogenic disturbances also may produce somatizations in which there are objective evidences of pathologic disturbance in the afflicted organ These clinical manifestations formerly constituted a no man s land Psychiatrists avoided trespassing upon the fields of organic disease Organicists in clinical medicine treated the local manifestation neglecting the psychogenic overtone Each patient became a battleground of divergent medical opinion Thus the patient with peptic ulcer was recognized as suffering from emotional instability Often the onset of gastric symptoms was directly related to a psychic

Regression—In the regression the libido having developed along normal lines returns or reverts to an earlier point of fixation These situations of greater gravity occur in every neurosis but particularly the compulsive and obsessive neuroses psychasthenia and the perversions The prognosis of regressions is graver and treatment less satisfactory

Compulsion—The compulsion constitutes an irrepressible urge to carry out what is apparently a needless and purposeless activity Thus the sufferer washes his hands indefinitely turns off lights carries out repetitive rituals involving unnecessarily strict attention to orderliness and tidiness and is occupied with all manner of superstitions and fetishes There is the necessity to touch wood avoid or walk on cracks in the pavement carry a lucky piece or charm and observe certain curious superstitions and rituals In more extreme degrees the patient develops such manias as kleptomania (stealing) toxicomania (compulsion to take medicine) dipsomania (the compulsion to drink) pyromania (compulsion to set fire) and trichotillomania (compulsion to pull out hair)

Perversion—The perversions are compulsions towards some abnormal form of instinctual gratification Often there is any one or more of the following sexual disturbances continued and chronic masturbation masochism sadism exhibitionalism or homosexuality Perverts indulge in such practices as pederasty cunnilinguism fellatio bestiality incest and necrophilia See pp 2400 2491

Displacement Projection and Rationalization—More complicated disturbances of the libido result in *displacement* and *projection* The former indicates a detachment of emotions from the objectionable idea and an attachment to some other less obvious or less objectionable object or person Thus the child with an unconscious homosexual urge develops a crush on a teacher of the same sex Projection implies that the repressed idea is regarded as belonging to another person Displacement and projection are used constantly by every human being in the formation of dreams and fantasies They become the instruments of illness when they begin to cause overt difficulties and are most commonly seen in the psychoses *Rationalization* consists in utilizing the unconscious in order to give plausibility to incongruous ideas

Dreams and Slips of the Tongue—Beside clinical symptoms Freud believes that neurotic conflict reflects itself in dream life and in slips of the tongue In the former the disturbance dramatizes itself through symbolization of a bewildering character In the course of psychoanalytic therapy the significance of these symbols and the seemingly innocent lapses in speech permit the trained observer to interpret their significance in terms of individual conflict

RESOLUTION OF THE NEUROSIS

It is inevitable that each life should involve conflict The manifestation may be latent useful or harmful

The Latent Neurosis—In the vast majority of individuals the conflict never becomes clarified It exists often like a birthmark as an accepted character trait or at worst as a nuisance which annoys the patient but does not seriously interfere with his life Thus an anxiety or a phobia per

PSYCHOSOMATIC DISTURBANCES

The Digestive Tract
Cardiospasm
Anorexia nervosa
Plummer Vinson syndrome
Peptic ulcer
Regional ileitis
Nonspecific chronic ulcerative colitis
Mucous colitis

The Circulatory System
Essential hypertension
Neurocirculatory asthenia (soldier's heart effort syndrome)
The arrhythmias

The Tegumentary System
Urticaria
Angioneurotic edema
Neurodermatitis
Acroparesthesia
Acrocyanosis
Erythromelalgia
Dermatitis factitia
Infantile eczema
Alopecia

The Respiratory System
Vasomotor rhinitis
Bronchial asthma

The Urinary System
Enuresis

The Ductless Glands
Hyperthyroidism
Simmonds disease
Diabetes mellitus
Hyperinsulinism
Obesity

The Gonads
Dysmenorrhea
Amenorrhea
Premenstrual tension
Frigidity
Impotence

The Voluntary Nervous System
Migraine
Epilepsy
Asthenopia

Psychic and Emotional Disturbances —Conversion symptoms may involve psyche and emotions Not infrequently patients develop automatism amnesia catalepsy trances clouding of consciousness lapses in consciousness narcolepsy somnambulism fugues delirium dream states stupor dual personalities and particularly disturbances in sexuality such as frigidity impotence nymphomania and satyrism Hysterical convulsions may assume the *passionate attitudes* mimicking sexual acts Abnormal moods occur as the *Ganser syndrome* in prisoners

Fixations and Regressions —Besides conflict and conversion Freud recognizes mechanisms of fixation and regression the clarification of which requires a preliminary statement of his views relative to the sexual instinct

The Development of the Sexual Instinct —Freud believes that the sexual instinct initiates at the beginning of extra uterine life and reaches a first culminating point (*early period*) at or before the fifth year The early period is followed by a *period of latency* in which the sexual instinct is inhibited or interrupted until the age of puberty when there is a *second climax* of development until normal gratification occurs Freud holds that the experiences of the early period of childhood in combination with the inherited sexual constitution form the disposition for the subsequent development of the individual character or the manifestations of his neuroticism

Repression and Fixation —When the sexual instinct *is* repressed the libido becomes arrested at some stage of infantile development The arrest of the libido in the early stage of development is termed a *fixation* Since masturbation and auto erotism characterize the infantile state the neurotic manifests these types of reaction and an infantile narcissism with preoccupation referable to oral and anal zones

The Anxiety Neurosis

There is a certain amount of anxiety that accompanies each medical condition and which persists throughout the course of the affliction The realm of the anxiety neurosis however is approached when the amount or duration of the anxiety is disproportionate to the cause when it dominates the clinical symptomatology interferes with the patient s well being and progress and impedes favorable progress

Clinical Manifestations —The anxiety neurosis is the commonest of all clinical derangements It may be acute approaching a panic state and it may be chronic or recurrent Anxiety symptoms may bear a repetitive pattern and the patient recurrently suffers from cancer blood poisoning lockjaw apoplexy or acute indigestion More often the panic states run the gamut from kidnapping through murder suicide malignant disease or grave infection

Provocative Cause —The anxiety may be precipitated by a tangible symptom or physical finding or it may result from a disturbing dream or from seemingly no cause whatsoever The patient with headache develops anxiety about a brain tumor meningitis or encephalitis A scratch causes fear of septic blood poisoning or tetanus a sebaceous cyst is regarded as cancerous and precordial pain as indicative of coronary thrombosis

At times the anxiety is unassociated with anything tangible and is based on *projections into the future* The housewife whose husband is delayed anticipates that he has been killed and that her child who has lingered on the way home from school has been kidnapped Each occurrence no matter how trivial or inconsequential has an affect of sufficient intensity to warrant the assumption that a major tragedy has occurred

Anxieties occur as vividly in *dreams* as in reality situations Characteristic of dreams is the *pavor nocturnus* in which the patient wakens suddenly in the midst of sleep with overwhelming and distressing anxiety symptoms Often the provocative dream is forgotten or repressed At times the panic state arises without apparent provocation in conscious cerebration or dream life In all likelihood many of these situations are conditioned by the sound of a voice an odor or a visual or tactile sensation repressed from consciousness

Autonomic Imbalance —The anxiety neurosis rarely occurs without accompanying disturbances in the realm of the psyche and the involuntary nervous system The patient almost invariably has other emotional difficulties and is usually troubled by a sense of insecurity and inadequacy especially as compared to self inflicted overconscientious standards More disturbing for the diagnostician are the autonomic accompaniments of anxiety since they *simulate organic disease* The acute panic is accompanied by tachycardia sweating pallor flushing shortness of breath feelings of suffocation choking sensations urgency and frequency of urination or explosive defecation The visceral manifestations of the autonomic imbalance often suggest that some acute circulatory accident or shock producing phenomenon has occurred

Reactions to Reality Situations —In striking contrast to the reaction of the patient to the anxiety producing mechanism is the stoical approach to reality situations When the sufferer from an anxiety neurosis is con

sists throughout the life span of most individuals and is relatively unimportant in the full ledger of the years

The Useful Neurosis —In some individuals the neurosis is of positive importance and may be necessary and useful It is the drive behind many great artists musicians writers physicians surgeons and political figures It was undoubtedly to this that Oliver Wendell Holmes referred when he stated that it was the cranks that make the wheels go round In contrast to the socially useful neuroses are the secondary gains which the neurotic wins at the expense of the rest of the community The woman with an hysterical headache or vomiting attacks receives a great deal more attention than her more integrated sister She may be coddled and protected by her family whereas otherwise she might have to assume additional and onerous duties in relative obscurity The neurosis is useful when it can be exploited to the advantage of the possessor The comedian who poses as a neglected unloved tramp is actually parading his neurosis for lucrative emoluments at the box office whether or not this is his deliberate intent We laugh and joke at references to impotence satyrism nymphomania or homosexuality We observe in the arts the delineation of the neurosis in the paintings choreography and the utterances of the artists In these examples the neurosis furnishes a method of expression and often a livelihood for the afflicted In a less obvious fashion the prosaic neurotic utilizes his neurosis through the mechanisms of *sublimation* and *rationalization* The anxious and insecure individual overcompensates and through excessive diligence and application accomplishes stupendous tasks The patient with an obsession or a compulsion becomes a slave to a routine to which he adheres come what may In other instances the drive is sublimated The tippler becomes a reformer the woman of easy virtue a prude and the prevaricator the watchdog of veracity

The Clinical Neurosis —In a relatively small number of individuals the neurosis is neither latent nor useful It interferes with life liberty and the pursuit of happiness It impinges upon the domain of clinical medicine and requires treatment The remainder of this chapter will deal with the clinical neuroses

CLINICAL VARIETIES OF NEUROSES

Before the clarification by Freud of the mechanisms of the neuroses certain descriptive disturbances were recognized and named though the labeling accomplished little purpose other than classification In light of newer developments it becomes apparent that certain of these categories deserve preservation since they afford diagnostic prognostic and therapeutic implications of considerable value

For present purposes the neuroses will be listed in the following manner

Anxiety neuroses	Traumatic neuroses
Neurasthenia	Compensation neuroses
Hypochondria	Compulsion neuroses
Conversion hysteria	Perversions
Anxiety hysteria	Mixed neuroses

reading and the differentiation reverts back to clinical judgment and a therapeutic test with iodine (p 1218)

If there is the slightest doubt concerning the integrity of the circulatory system the most elaborate laboratory examinations are indicated for the mutual protection of patient and physician An electrocardiogram may reveal surprising changes in either direction When there is reasonable doubt and a probability that organic circulatory disease is present or is accompanying the anxiety it is wiser to suspend judgment and wait upon observation for several hours or even days

Course —The anxiety neurosis may be wholly latent It may be provoked on occasions by all manner of nonspecific stimuli such as trauma illness war deprivation the death of a relative or friend pregnancy the menopause a fire or an accident The course may be more or less chronic with such persistent anxiety as to exhaust the good will patience and affection of all members of the immediate family household or social group Anxiety also may be an early symptom of the major psychoses and in the face of doubt observation by a psychiatrist is advisable

Treatment —Many anxiety neuroses are sufficiently mild so that intelligent supportive treatment by the practitioner makes it possible for the patient to live with his neurosis In any event nontechnical psychotherapy is valuable as a therapeutic test with referral to the specialist if there is not a material reduction in the symptomatic difficulties within a reasonable period of time

Reassurance —*The elements which enter into a psychotherapeutic result* are many and varied The most potent factor is that of reassurance after the completion of the diagnostic survey with particular focus on the area indicated by the symptoms Reassurance is accompanied by the positive assertion that the practitioner regards the complaint as genuine and not as an imaginary figment or an exaggeration The wise physician will stress this point he will not deny the existence of the symptom jeer at it or indicate that the patient is overstating the degree of discomfort Often it is a great comfort to the patient to have the practitioner report that he himself has suffered from the same complaint and testify to the intensity of the distress

Prognosis —Reassurance *is fortified by* the promise that the symptoms will abate and the prediction that they will recur on provocation If the latter precaution is not taken the confidence of the patient in his therapist may be rudely shattered upon exacerbation of the derangement

Observation of Acute Episodes —The first interview relative to the condition is terminated by the request that the physician be notified in the event of an acute episode Particularly in panic states the patient may accept the fact that he is normal at the time of the examination but will insist that something quite different must transpire during the acute phase such as the *pavor nocturnus* The physician extracts the promise that the patient will hasten to the office immediately the symptoms are noted so that an examination may *be made in the midst of the occurrence*

Follow up Visits —No matter how greatly the patient seems relieved after the first interview a series of follow up visits is planned If the patient seems unwilling to come for an interview it is justifiable to give a hypodermic injection of any bland substance such as neurophosphate a

fronted with a truly serious emergency such as an operative procedure he may respond with courage and steadfastness which amazes the practitioner who has anticipated all manner of annoying and disturbing psychic and emotional overplay

Simple and Complicated Anxiety Neuroses —The anxiety neurosis exists in a *simple* form when it is *unaccompanied by organic disease* An important problem in differential diagnosis arises when the anxiety accompanies a *definitive physical finding*, such as a hypertension, valvulitis glycosuria or albuminuria Under these circumstances it is the function of the practitioner to evaluate the proportion of the distress resulting from the organic finding and that which is derived from the aura of anxiety

Still more difficulty arises when the patient with a known anxiety neurosis develops *acute symptoms* such as pain in the abdomen The practitioner who knows his patient well discounts a certain proportion of the reported complaint as a manifestation of the anxiety neurosis In the back of his mind however he must remember that the patient with the neurosis as well as the normal integrated individual may develop acute appendicitis incarceration of a hernia or perforation of a peptic ulcer There is probably no greater dilemma in the practice of medicine than that of attempting rightly to assign the significance of the symptoms in a case of anxiety neurosis On the one hand the conscientious physician fears to give the symptoms the weight proportionate to the degree of complaint for under these circumstances unnecessary operative procedures may be initiated Opposed to this if the symptomatology is underrated the patient with the anxiety neurosis cries wolf and an inflamed appendix goes onto perforation merely because the overcautious physician has refused to accept the reported symptoms at face value

Pathogenesis of Anxiety Neurosis —The anxiety neurosis is seen throughout life but is most frequent in relatively young patients often of adolescent age The mechanism according to the Freudians is a conflict that is usually related to a healthy sexual appetite without the means for gratification Oftentimes as the analytic school has particularly pointed out the anxiety is in reality the expression of wish or an unsuccessful effort at the attainment of a purpose

Diagnosis —The diagnosis of the anxiety neurosis is made on clinical grounds its positive affirmation does not preclude the possibility of the presence of concomitant organic disease No matter how obvious may be the psychogenic nature of the disturbance both for diagnostic and therapeutic purposes the practitioner is under obligation to perform a *meticulous physical examination* with particular reference to the area organ or tissue to which the symptom is referred The fact that the patient has a neurosis does not eliminate the possibility that he also has a cancer or tuberculosis In the acute panic states the problem is of serious complexity particularly if the symptoms are referred to the circulatory apparatus since the anxiety in and of itself may produce tachycardia elevation of blood pressure and symptoms suggesting peripheral vascular failure (shock)

In the chronic anxiety states there is often a resemblance to *hyperthyroidism* If possible a basal metabolic estimation is performed but often by the very nature of the neurosis it is difficult to get an accurate

thenic may develop superimposed chronic disease such as active tuberculosis carcinomatosis or a blood dyscrasia

Many of the bitterest experiences of clinical practice arise from dealings with neurasthenics Tiring of their old physician they seek a new face or an institutional work up and report in triumph that there has been found a new physical sign such as low blood pressure a fallen womb or a ptosed kidney which explains the symptoms previously dismissed as neurotic With facile ignorance of the total situation the new physician gives symptomatic therapy or performs an operative procedure such as a gynecologic plastic a nephropexy or an appendicectomy For a while a miracle is reported primarily due to bed rest but with recurrence of symptoms the situation is intensified The passage of time reveals the error to the patient but does not repair the injury to the reputation of the original medical advisor

Pathogenesis—Freud s original explanation of neurasthenia was that of an organic neurosis which he attributed to actual debility resulting from excessive sexual activity Later analytic interpretations linked it closer to the mechanisms of the schizophrenic process

The neurasthenic often has suffered from lack of affection and frustration in early life As a child he was unloved unwanted unattractive and unpopular Prolonged and frequent masturbation may represent a compensation for the inattentiveness of others and the inadequacies of parental love

Course—The symptoms of neurasthenia do not usually become manifest until adult life at which time they are precipitated by some life situation such as a disappointment in love or work The younger neurasthenics are almost all early schizophrenics The symptoms are more or less chronic and acute episodes such as characterize the anxiety states are rare

Diagnosis—Since many chronic diseases begin with fatigability and the symptoms of neurasthenia the practitioner refrains from making the diagnosis of the neurosis by any method other than by *exclusion* Early tuberculosis hyperthyroidism diabetes mellitus the blood dyscrasias brucellosis deep seated malignancy the anemias the avitaminoses and adrenal cortical deficiency may begin with identical symptoms The patient is entitled to a complete blood examination urinanalysis the recording of a temperature graph a chest film an estimation of the basal metabolic rate a tuberculin skin test and repeated physical examinations before a definite commitment is enunciated

Since cachexias and chronic infections may arise in the neurasthenic just as in the integrated individual physical examination is repeated at intervals to ascertain that organic complications have not been obscured by the psychogenic manifestations

Treatment—The treatment of neurasthenia is conducted according to the principles of therapy used in the anxiety neurosis (p 1347) Additionally attempts are made to stimulate mind and body

After the patient is assured as to his organic integrity he is told of the presence of positive physical findings such as a low blood pressure a subnormal temperature or a slight anemia Women are made aware of nephroptosis or such gynecological abnormalities as uterine malpositions It is carefully explained that none of these deviations from the normal is

vitamin preparation or distilled water The injection visits give the physician an opportunity to renew his psychotherapeutic attempts The patient often begins to unburden himself of his problems and accomplishes a healthy ventilation The material uncovered affords an opportunity to the practitioner to make interpretations and explanations of difficulties and perhaps link them with the symptoms

At each visit it is wise to reexamine the organ or tissue to which the symptom is referred so that further reassurance is given Meanwhile in a seemingly offhand manner it is often possible to direct the play hobbies recreation studies and reading and the adjustments of the patient to his associates family and environment

As soon as the patient develops an understanding of his difficulties and the anxieties abate the hypodermic injections are terminated but the patient is not discharged He is instructed to attempt to get along on his own momentum but the promise is exacted that upon a return of the symptoms he will resume therapy

Drug Therapy—Psychotherapy is abetted by the use of a *simple sedative* such as phenobarbital 0 015 gm (¼ grain) after meals and a *hypnotic* in average doses at bedtime

Reference to Specialist—The patient with resistant symptoms or prepsychotic manifestations is referred to the psychiatrist for more complete study and observation Provided there is no total personality defect psychoanalysis gives promise of brilliant therapeutic result in obstinate anxieties for more rapid results the hypnagogic reverie (p 1328) may be tried

Neurasthenia

The symptom complex of neurasthenia is characterized by excessive mental and physical fatigability irascibility a tendency to depression cardiac irritability headache sexual difficulties and gastro intestinal symptoms In general the neurasthenic is a discouraged and disappointed person Little pleases him and nothing is truly satisfying His sexual life is especially annoying for either the libido is lacking or potency is impaired The female is usually frigid or at best lacking in normal sexual response Almost invariably in neurasthenia there is a problem of masturbation This habit has usually persisted into adult life and is associated with considerable shame and a feeling of guilt In fact the feelings of weakness and irritability are usually attributed to masturbation

Examination of the neurasthenic often reveals a subnormal temperature a low blood pressure a slow pulse rate and a lowering of the basal metabolic rate without clinical evidences of hypothyroidism

Simple and Complicated Neurasthenia—In *simple* neurasthenia there is no insurmountable difficulty in eliminating the possible presence of organic disease *Complicated* neurasthenia is that in which there is a superimposed and unrelated pathologic change or in which organic disease develops in the patient who previously suffered from simple neurasthenia

As in the anxiety neurosis the practitioner must guard against diagnostic pitfalls He is called upon to evaluate the symptomatology in the neurasthenic who has also a healed tuberculosis at an apex a mild anemia a persistent hypotension some albuminuria or glycosuria His knowledge of his patient s neurosis must not obscure the possibility that the neuras

assistance of massage graded exercise and water cures occasionally achieves results that cannot be obtained at home See p 3764

Formal Psychotherapy—Formal psychotherapy is most disappointing in the treatment of neurasthenia Unless attempted early in the course of the process even an adequately performed psychoanalysis has no brilliant prospect of success Shock treatment may be tried in desperation with little promise of more than passing benefit

HYPOCHONDRIASIS

Hypochondriasis is probably not a separate neurosis but is more likely a *neurotic symptom encountered in the setting of almost any other neurosis* It consists of an exaggerated preoccupation with the body and its functions It may or may not be associated with fear and anxiety To the hypochondriac each deviation in function and feeling assumes the proportion and significance of a major illness until the neurosis occupies the main interest and concern of the patient s total energy

Pathogenesis—The hypochondriacal patient is consumed with an interest in his own body (*narcissism*) and apparently enjoys the punishment of the emotions set up by the hypochondriacal response (*masochism*) *The neurosis is associated with insecurity and a feeling of rejection* In a sense the preoccupation with one s own body represents an *attempted compensation for the lack of interest by others*

Diagnosis—The hypochondriac is entitled to a consultation with the specialist psychiatrist since the symptoms may be the early manifestations of *schizophrenia* or the depressed stage of a *manic depressive psychosis*

Treatment—An attempt may be made by the practitioner to treat hypochondriasis in the manner of neurasthenia (p 1350) After a reasonable effort and almost inevitable discouragement the patient is encouraged to apply for formal psychoanalytical therapy since less drastic measures offer little hope of a satisfactory result Shock treatment may be attempted as a measure of desperation in a probatory fashion

CONVERSION OR TRANSFERENCE NEUROSES (HYSTERIA)

The Freudian concept of hysteria involves conflict between personality *and a wish that is out of harmony with the ego and is repressed* The repression is unsuccessful and the unconscious wish being dynamic forces its way into consciousness in a disguised symbolic form The resultant hysterical symptom is thus a compromise

The term conversion hysteria coined by Freud designates the phenomenon whereby emotional conflicts are referred to or projected as bodily symptoms An anxiety hysteria exists when the conversion mechanism has an additional component of fear

The Hysterical Personality—The hysterical personality makes the impression of an immature individual Emotional affects are free easily stimulated and histrionic although psychosexual development may be full and complete The hysteric is usually shy and reserved but given to intense demonstrativeness There is an obvious contrast between the shal

the cause or the result of the neurosis If this precaution is not taken the next examiner may seize on the tangible evidence as a 'pathogenetic straw' with a resultant exploitation of the patient and condemnation of the better informed physician It would be impossible to estimate the number of unnecessary operative procedures, particularly in the field of gynecology, that have been performed for the relief of symptoms due to neurasthenia The temporary benefits of these adventures into surgery are the delight of those who report short term results but they are the despair of the 'compleat practitioner' to whom the patient eventually returns with all of the pristine disturbances

The neurasthenic is stimulated to more intense effort at work play hobbies or recreations It may be possible to remotivate efforts and sublimate them in some useful direction Often a neglected neurasthenic responds with almost pathetic eagerness to the normal amount of interest and kindliness

Drug Therapy—Repeated visits are assured by the administration of some type of hypodermic injection One of the commonest forms of injection therapy consists of the use of the so called "neurasthenic serum" which is nothing other than glycerophosphates with a modicum of cacodylate of soda Enthusiasts for vitamin and hormone therapy give mixtures of the soluble components of B complex or adrenal cortical substance The latter is particularly indicated by the resemblance of some of the symptoms of neurasthenia to Addison's disease A simpler and less expensive approach to the same end is accomplished by advising the ingestion of sodium chloride in daily doses of 3 to 6 gm Unless the patient or his physician notes a seemingly specific improvement from hypodermic injections this form of therapy is discontinued as soon as possible

The hypodermic injections are then supplemented by the oral administration of *amphetamine sulfate* (benzedrine) using 2 5 or 5 mg ($\frac{1}{24}$ to $\frac{1}{12}$ gr) each morning on arising and again at noon time provided that nocturnal sleep is not disturbed While benzedrine is being administered a *hypnotic* (*q v*) is ordered at bedtime to assure adequate sleep

The patient with a low basal metabolic rate receives corrective doses of *thyroid extract* (p 1189) When the level of oxygen consumption approaches the normal the physician decides on the basis of the patient's subjective response whether substitution therapy should or should not be continued In the event of reasonable doubt the drug is stopped for a trial period of three weeks in order to obtain more conclusive information

Each form of drug therapy has advocates and occasional success By and large the lack of consistency and the numbers of the "specifics" lead to the inescapable suspicion that the common denominator for good is the faith of the patient and the suggestion that is practiced purposefully or subconsciously by the therapist Under no circumstance is a permanent result anticipated and the patient often must resolve to endure the symptoms and live within the limit of his capacities

Rest Cures—At times the practitioner despairing of other forms of therapy attempts a *Weir Mitchell* "*rest cure*" often with surprisingly good results Naturally the effects are not permanent and upon discharge the patient is warned that a recurrence is to be anticipated under which circumstance the "rest cure" is repeated *Spa therapy* with the adjuvant

whereas in anxiety hysteria the anxiety breaks through Similarly in a *hysterical depression* the depression breaks through the symptomatic expression or the patient alternates between the neurotic mood and the conversion process In true conversion hysteria the patient is indifferent to his illness whereas in the mood disturbance there is suffering and a desire for treatment

The patient who develops anxiety hysteria is usually immature and suffers a great mistrust of other people There may be a surface friendliness but it is a veneer that does not stand the test of conflict Often the anxiety hysteric has been a spoiled child of unreliable and undependable parents who has been given excessive adoration in brief moments and long periods of neglect Usually material gifts have been offered instead of continued emotional security As a result these children suffer from rages and tantrums They enjoy an extravagant fantasy life along romantic and erotic lines This is often reflected in their dress which is gaudy with an excessive use of cosmetics and an assumption of seductive behavior The outward appearance usually cloaks a lack of experience or actual frigidity

Pathogenesis—Conversion hysteria is a reaction adopted unconsciously for a very definite purpose It serves to disguise some problem frequently the satisfaction of a wish whose conscious gratification cannot be permitted The conflict that arises is converted from an emotional difficulty into a bodily symptom Thus the Freudian explanation reveals the purposefulness of the symptom despite the fact that the patient is consciously unaware of its function It explains the unconscious gratification that the patient derives from his symptom and the marked contrast which exists between the seeming shallowness of the emotions and the intensity and dramatic manner in which they are expressed

Diagnosis—The possibility of conversion hysteria is the lurking figure that haunts every diagnostic problem The astute practitioner can only avoid serious difficulties by positive exclusion of every other possibility Nor should he fall into the error of interpreting as hysterical every disturbance occurring in a hysterical individual The sufferer from hysteria is as apt to develop acute appendicitis organic cardiac disease tuberculosis or carcinoma as his emotionally normal fellow

In his dealings with the hysterical personality the practitioner should maintain a high index of suspicion relative to conversion symptoms but he should not take the step between suspicion and definitive diagnostic commitment until he has exhausted all physical and laboratory methods of investigation Consultants are used freely especially when there is the possibility of a surgical condition

Treatment—Since the manifestations of hysteria are characterized by sudden onset and sudden dissipation treatment by almost any means is capable of apparent success Hysterical patients are the prize of all charlatans and fakirs within and without the medical profession They are relieved of their symptoms by suggestion faith hypnosis religion yoga massage glands vitamins hypodermic injections electrotherapy the actual cautery pink pills bizarre diets incantations Voodooism and modalities of similar ilk

The suggestibility of the patient arouses a state of alertness in the conscientious practitioner He recognizes that almost any element in

lowness of the emotions and the intensity and theatrical effects with which they are expressed The patient in an hysterical outburst, simulate the actor whose burst of passion turns off like a light when the curtain of the drama descends It soon becomes apparent that emotions are mostly spurious and feigned

The hysterical personality is usually capable of only brief personal attachments More normal individuals tire of the drama that has repetitiveness and intermissions but no final curtain

Clinical Manifestations of Conversion Hysteria—Conversion hysteria is the least severe and the most easily curable of the neuroses The conflict usually appears at a later period of childhood The symptoms arise suddenly and dramatically and often leave in similar fashion Actually however the underlying tendency to conversion is quite as obstinate as any other basic neurotic mechanism with the consequence that on provocation the patient is likely to flare up with other conversion symptoms

The variety of the conversion symptoms have been previously noted (p 1341) They run the entire gamut of the subjective complaints and objective manifestations known to clinical medicine They include sensory motor autonomic visceral psychic and emotional manifestations

The ***sensory complaints*** include pains such as headache coccygodynia mastodynia and glossodynia paresthesias may be pruritic particularly in erogenous zones such as the vulva vagina rectum and anus and there may be anesthesia hemianesthesia anosmia amaurosis myopia mutism and the like ***Motor abnormalities*** take the form of spasms or paralyses The repertoire includes tics tremors contractures paralyses stammering stuttering and aphonia

The ***autonomic manifestations*** may be vasodermal cutaneous or referable to the functional disturbances in the structures containing smooth muscles There are thus produced gastric and intestinal neuroses globus hystericus aerophagia cardiospasm anorexia nervosa bulimia pica nervous vomiting attacks of diarrhea or obstinate constipation vaginismus peroneal spasm urinary retention instability of pulse rate and rhythm hypertension hypotension lability of the blood pressure episodes of tachycardia and cardiac irregularity peripheral vascular spasms sighing and gasping respirations bronchospasm and the like With severe autonomic imbalance or protracted smooth muscle and glandular disturbances *somatization* results and organic disease is produced as an end result of the conversion hysteria The complete list of the *psychosomatic manifestations* is elsewhere given (p 1344) but the commoner syndromes include peptic ulcer essential hypertension and neurodermatitis

Finally the hysterical individual may develop *psychic* and *emotional* disturbances which simulate the psychoses They may involve perception (p 1292) consciousness (p 1293), attention (p 1296), thought production content and progression (p 1297) memory (p 1298) affect (p 1300) appetite (p 1302) sexual behavior (p 1298) sexual function (p 1304) sleep (p 1304) motor behavior (p 1308) speech (p 1310) and posture (p 1310)

Clinical Manifestations of Anxiety Hysteria—An anxiety hysteria is merely a conversion hysteria with an additional symptomatic anxiety In the true conversion hysteria the patient has masked completely all affects

Compensation Neuroses

There is a high incidence of neuroses which follow trifling accident or injuries when the patient receives compensation from an insurance company or from the state The symptoms include the entire battery of neurotic difficulties anxiety states and symptoms simulating conversion hysteria The conditions have in common a striking disparity between the minimal injury and the maximal amount of emotional involvement The differentiation of a traumatic neurosis from malingering may tax the skill of the specialist to whom the problem is referred particularly in view of the litigious possibilities

Perhaps the most important aspect of these conditions is the basic postulate that no traumatic neurosis can be successfully treated as long as the patient is receiving compensation It is vastly to the patient s advantage that *he receive a lump settlement pay for his own therapy* and make his adjustment within the framework of his compensation

Neuroses of Regression and Perversion

The neuroses of regression and perversion represent fixations at infantile levels They are difficult of treatment but fortunately much less common than the simple and conversion neuroses

Clinical Manifestations of the Compulsion Neuroses—Compulsive acts of significant degree are fortunately uncommon They represent an intrusive need to act or think in a definite repetitive manner despite the intellectual awareness of the uselessness of the act or thought of its disagreeable or painful nature The patient is compelled to think or act in a particular manner despite reason will power determination or a sense of humor

Many patients have routines of behavior to which they cling tenaciously and without which they feel uncomfortable They have certain rituals in arranging clothes pillows and chairs They must go to bed make love attend to their bodily needs conduct their businesses and manage their households in a set fashion despite the inroads that their conduct makes upon their own or their associate s comfort convenience and economy Occasionally the compulsive idea takes on the nature of a religious ceremonial or a superstition such as touching wood stepping on the cracks *in the pavement or avoiding the cracks in the pavement* At times it becomes necessary to count numbers count windows the number of steps or the number of cars in a train The obsession may be relative to health and require repeated washing of the hands violent daily catharsis repetition of enemas and colon irrigations or an aversion to shaking hands because of fear of soiling and contamination There may be doubts concerning the locking of a door the closing of a window or the turning out of the lights and gas jets Much more serious are the compulsions relative to jumping off a height plunging a knife into flesh or the impulse to dive under approaching trains

In addition to these compulsive acts are compulsions in thinking in which the train of thought cannot be interrupted and must proceed along certain lines (*idee fixe*) The compulsion or obsession may take on antisocial manifestations such as kleptomania pyromania dipsomania toxicomania or trichotillomania

which the patient has faith will produce relief of symptoms and he does not wish to be misled by his apparent therapeutic triumph He knows that the symptoms will recur on provocation and that the patient who elaborately sang his praises and flattered his ego as of the current week may with equal fervor and enthusiasm brand him as an ignoramus and fakir and start a campaign to have his license taken away before the sun sets on another day

Conscious of the volatility of his patient the practitioner proceeds with caution and timidity He reassures his hysterical patient that there is no evidence of organic disease and follows the principles laid down in the management of the anxiety neurosis (p 1347) He exerts even greater care to avoid any implication that the disease is imaginary the symptoms exaggerated or the condition hysterical After several sessions for which the patient returns for hypodermic injection or the application of some physical modality the psychotherapeutic approaches assume increasing prominence and the tangible modalities are dropped *Sedatives* and *hypnotics* often assist in the progress of events When the confidence of the patient has been obtained and the symptoms begin to abate or entirely disappear the superficial conflicts become apparent through the patient s free ventilation

As soon as the symptoms lessen it is wise to warn of *recurrences* and *exacerbations* on provocation and then is the time to intimate that there is a relationship between psychic conflict and clinical disturbance The patient being more or less educated to this thought through the course of previous conversations resents the implication less acutely and may even leap at the explanation In more favorable instances adjustments remotivations and sublimations may terminate the problem

The patient who has refractory manifestations recurrences or possible psychotic personality difficulties is referred to the trained psychiatrist Again almost any type of psychotherapy will yield a favorable result but the more permanent and complete effects are to be anticipated from the psychoanalytic approach Shock therapy is ill advised but hypnosis and the hypnagogic reverie are worthy of trial under expert supervision

Traumatic Neuroses

The traumatic neuroses are usually included in the group of hysterical disorders They usually occur after events in civil or military life which are interpreted by the individual as a sudden threat to his existence Kardiner has postulated that the traumatic neurosis develops as a protection against further hurt from a hostile world The patient looks upon himself as helpless in this world and may revert to childlike mechanisms in the service of this need for defense

The milder manifestations include irritability excessive sweating trembling dizzy spells tics nausea and vomiting There may be confusional states fugues and isolated conversion phenomena Quite characteristic of the traumatic neuroses are terrifying dreams which repeat the provocative experience

The *treatment* of traumatic neurosis is best left to the specialist who requires an extraordinary degree of skill and much experience

serious psychoses and this decision is *specialist* province The differential diagnosis of neurosis versus psychosis is elsewhere delineated (p 1313)

Treatment—The treatment of compulsion states and perversions is difficult and unrewarding A *psychoanalytic* approach may be of considerable value if the condition is not of excessively long duration so that the entire personality has become involved in a rigid obsessional pattern

Shock therapy has been used with some degree of success but is not employed unless the incapacity justifies the risks of the procedure Examples of conditions which warrant shock therapy are the manias which have legal implications such as kleptomania pyromania or compulsive exhibitionism and the perversions In desperation prefrontal lobotomy may be attempted

Mixed Neuroses

It is unusual for any neurosis to conform strictly to any of the patterns previously described Even the well integrated physician recognize that he himself reflects anxieties fears compulsions obsessions and mild conversions He knows too that normal sex life is associated with occasional furtive dallyings with the mechanisms of perversion The neurotic also presents a galaxy of clinical phenomena Rarely do these reaction pictures fit into any definitive pattern and in a sense all neuroses are mixed The exact classification which has some prognostic and therapeutic implications rests upon the dominant presenting derangement

DISORDERS OF TOTAL PERSONALITY

The disorders of total personality include the *abnormal conduct and behavior patterns* as well as the *psychoses* These in contrast to the partial reaction of the neurosis are total personality disturbances The minor manifestations are the province of the practitioner but the management of the psychoses enters the province of the specialist often with medico legal implications

Personality Behavior and Conduct Disorders

The individual personality and behavior pattern varies according to many characteristics which include physical qualifications intellectual and educational attainments emotional social moral and spiritual components and hereditary acquired financial ethical and political influences

The practitioner learns to expect more of a husky healthy adult than of a puny sickly child The patient who has had educational advantages ought to be more reasonable and tractable than the illiterate though this is not always in accordance with experience Varying degrees of conduct are anticipated in individuals brought up under varying social circumstances The child reared in the home of the lord of the manor is expected to demonstrate more of the social graces than a field hand whose habitat is the barn though this may not necessarily be the case Moral and spiritual values vary in different communities and in different portions of the same community and each is judged by the surroundings in which he was cultivated

In the majority of instances the compulsions are mere habits and it is doubtful whether any one of us is completely free from at least one manifestation It is when the resistance to the obsessional pattern causes extreme discomfort *interferes with daily routine or prevents functioning* where it is so bizarre as to be obtrusive and painful that the realm of clinical medicine is entered and symptomatic relief is sought Under these conditions the personality becomes dominated by the neurosis and failure to act in the compulsive manner is associated with severe superimposed anxiety

At times the compulsion is socially useful Under these circumstances the patient is scrupulously neat and orderly almost to a fault Hyperconscientiousness and shyness conservativeness and penuriousness miserliness and general conformance are in a final analysis compulsions While they seem to be social assets actually the individual who exhibits these traits is dependent and requires a good deal of reassurance and approval

Pathogenesis—The seeming lack of meaning and reason in the compulsive acts becomes clarified in the interpretation of the Freudians The original compulsive idea is a psychic phenomenon which has been elaborated as a defense against an early sex transgression The original objectionable idea containing a charged emotional content is repressed the affect is dissociated from the idea and attached to a harmless concept which is then permitted to enter consciousness A further defense against the repetition of the tabooed wish is the development of a ceremonial which is attached to the obsessive idea Though the compulsion seems completely dissociated from reason and the normal mental processes analysis establishes that the compulsive thought or action is actually a useful symbolic defensive mechanism of the unconscious It is necessary to the mental economy and serves as a *compromise gratification of infantile wishes* the obsession substitutes gratification for repressed guilt laden and feared desires

The neurotic uses his rigid attitudes and methods of reaction in an attempt to achieve a feeling of safety and security as though he would create a world so full of order and regulation as to protect himself from the problems and disorders of everyday life To accomplish this in the execution of the compulsion or obsession the individual may face danger humiliation jeering degradation and bodily injury

Clinical Manifestations of Perversion—The various deviations of sexuality, sexual behavior and sexual intercourse are elsewhere described They may serve in normal individuals as variations on a theme or as prologues or accompaniments to fornication in the natural manner When they dominate sexuality to the exclusion of normal relationships they enter the realm of the neuroses of regression and present profound clinical problems often complicated by disturbances due to shame guilt hostility, alcoholism and addiction

The perversions (p 2409) are franker fixations of infantile sexual development and are very resistant to treatment

Diagnosis—The diagnosis of compulsional neuroses and the perversions offers no difficulty relative to somatic medicine It is important however to ascertain that the manifestations are not herald symptoms of the more

Manifestations of Conduct Disorders —The conduct disorders created by personality difficulties and the psychopathic personality are legion They begin in childhood and end with senility They include crying spells tantrums by day and terror dreams by night failure to accomplish control of the urinary and defecation reflexes fears abnormal obstinacy lying *cruelty spitting up and vomiting of food day dreaming* lack of sociability truancy nail biting thumb sucking stammering stuttering insomnia cyclic vomiting anxiety phobias obsessions compulsions tics manifestations of hysteria *dulness backwardness maladjustments in social groups* and contacts misconduct in school overzealousness in conduct behavior and studies masturbation enuresis food capriciousness destructiveness sleep-walking conflict with fellow pupils or teachers and abnormal shyness

Adolescent personality problems include excessive emotional reactions relative to adventure and exploration worry egocentricity disturbance in sexuality such as excessive masturbation inordinate interest in the opposite sex inordinate shyness and retreat from the opposite sex crushes for individuals of the same sex running away from home or inadequate emancipation from parents and the household juvenile delinquency feelings of inferiority or superiority and the tragedy of adolescent suicide

In adult life the problems that are commonly encountered include lack of success or inordinate preoccupation with the career profession or business failure to marry or the accomplishment of a bad marriage sexual maladjustment in marriage either excessive insufficient or uncoordinated intercourse tendencies toward homosexuality and perversion child parent and parent child misunderstandings and maladjustments and parsimony and lavishness

Problems of the *aged* are most tragic The gaffer and his granny deteriorate interest in others is lost egocentricity is emphasized neatness and decency are replaced by untidiness soiling drooling and sexual exhibitionism by word and deed

Complications —The psychopathic personality tends to get into difficulties with all manner of antisocial conduct Criminal personalities delinquents vagrants prostitutes zealots hermits religious maniacs reformers *moralists super athletes sex perverts drug addicts* and *suicides* are recruited from within their ranks

Sexual Perversion —See p 2409

Drug Addiction —The common types of drug addiction include alcoholism morphinism cocainism the inordinate use of bromides barbiturates and hypnotics and the smoking of marihuana

Independent of the type of addiction the problem is essentially that of dealing with a personality deviation With relatively few exceptions the addict is a distorted personality It is the normal reaction to use *drugs when needed and to eschew them as soon as the indicated symptom* has abated It is for this reason that we have elsewhere stressed the psychological rather than the pharmacological approach to the treatment of alcoholism (p 3818) *morphine addiction* (p 3861) cocainism (p 3916) and habituation to sedatives and hynotics (p 3843) The marihuana problem is beyond the province of medicine since it constitutes a legal offense *and is to be handled by the proper authorities*

The assessment of standards is certainly not the business or the province of the practitioner However when dislocations occur which give rise to clinical symptomatology or interfere with the welfare security and peace of the patient and his environment, the problem is often deposited in the lap of the family doctor

Types of Disturbed and Psychopathic Personality—Deviations of personality are of great importance from the viewpoint of the individual and of society Patients may present a host of varying difficulties manifestations and symptoms These may be transitory or episodic or they may be chronic and persistent They may be accepted as manifestations of 'queerness" or 'eccentricity' under which circumstance they have mere nuisance value' or they may reach the point where they disturb the pursuit of a human life and harass household social and business associates

In general the personality disorders are characterized by *emotional insecurity* and *childishness* The patient is often unreliable and irresponsible Difficulties begin in childhood and continue with or without interruption throughout school years and the span of a life There seems to be no capacity to learn from experience Impulsive behavior without regard to the welfare and needs of others characterizes conduct Underneath a surface cleverness and glibness there is often bad judgment and a total lack of regard for truth and honesty

In the *neurotic behavior disorder* the neurotic mechanisms manifest themselves in disturbances in the mode of living with relatively few outspoken neurotic symptoms The *psychopathic behavior disorders* represent personality disturbances in which psychotic mechanisms disturb the general adjustment to life without the production of a fullblown psychosis The psychotic personality will cheat badger and steal without a sense of shame He frequently involves others in manipulations often with tragic consequences to innocent members of a family or their friends The psychopaths are notorious liars and purveyors of wild unreal schemes always with totally unwarranted expectations of success

Throughout these excesses these patients exhibit a poverty of assets They are incapable of lasting adult love relationships Their companions most often are like themselves in that they are selfish inadequate irresponsible and impulsive—all of which may be summarized by the terms *egocentric* and *narcissistic*

As *complications* of the psychopathic personality there result excesses in drinking in the use of tobacco and drugs sexual perversions and criminality Bad marriages and divorce mark and suicide frequently terminates their earthly careers Their roster includes perverts criminals tramps crackpots unsuccessful inventors and visionaries pathological liars eccentrics hoboes hermits and seekers after glory At times the psychopathic personality makes a virtue of his shortcoming and succeeds as an artist a religious or political reformer or as a leader of the rag tag and bobtail

Etiology—The etiology of personality disorders is not clearly defined The factors of heredity alcoholism poverty malnutrition and illegitimacy have been stressed As usual the organicists have sought the answer in pathologic changes such as might occur with inflammation particularly congenital syphilis but the evidence is meagre and unconv

Manifestations of Conduct Disorders —The conduct disorders created by personality difficulties and the psychopathic personality are legion They begin in childhood and end with senility They include crying spells tantrums by day and terror dreams by night failure to accomplish control of the urinary and defecation reflexes fears abnormal obstinacy lying cruelty spitting up and vomiting of food day dreaming lack of sociability truancy nail biting thumb sucking stammering stuttering insomnia cyclic vomiting anxiety phobias obsessions compulsions tics manifestations of hysteria dulness backwardness maladjustments in social groups and contacts misconduct in school overzealousness in conduct behavior and studies masturbation enuresis food capriciousness destructiveness sleep walking conflict with fellow pupils or teachers and abnormal shyness

Adolescent personality problems include excessive emotional reactions relative to adventure and exploration worry egocentricity disturbance in sexuality such as excessive masturbation inordinate interest in the opposite sex inordinate shyness and retreat from the opposite sex crushes for individuals of the same sex running away from home or inadequate emancipation from parents and the household juvenile delinquency feelings of inferiority or superiority and the tragedy of adolescent suicide

In adult life the problems that are commonly encountered include lack of success or inordinate preoccupation with the career profession or business failure to marry or the accomplishment of a bad marriage sexual maladjustment in marriage either excessive insufficient or uncoordinated intercourse tendencies toward homosexuality and perversion child parent and parent-child misunderstandings and maladjustments and parsimony and lavishness

Problems of the *aged* are most tragic The gaffer and his granny deteriorate interest in others is lost egocentricity is emphasized neatness and decency are replaced by untidiness soiling drooling and sexual exhibitionism by word and deed

Complications —The psychopathic personality tends to get into difficulties with all manner of antisocial conduct Criminal personalities delinquents vagrants prostitutes zealots hermits religious maniacs reformers moralists super athletes sex perverts drug addicts and suicides are recruited from within their ranks

Sexual Perversion —See p 2409

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While it is true that the practitioner rarely has expert knowledge of many of the personality disorders and their ramifications there is often no one else to whom the disturbed family group can go Under these circumstances there is no alternative but to make a determined and honest effort to accomplish a solution that promises the greatest prospect of benefit for the greatest number

Suicide—There has been an alarming increase in the incidence of self destruction The physician is required to protect his patient and himself by meticulous efforts at prophylaxis and by his refusal to fall into the commission of many common errors for which atonement is difficult Thus it is not true that (*1*) patients who threaten suicide will never make the attempt (*2*) the patient who has attempted suicide by one method is restricted to that device and if protected from this particular type of hara kiri is safe (*3*) the emotionally disturbed patient in a stage of recovery is safe from suicidal tendencies and need not be so carefully watched and (*4*) suicide is prevented by appeals to reason morality and fear of the hereafter

In his dealings with potential suicides the physician strives to make all of his mistakes on the side of conservatism It is his function to protect his patient and himself by demanding twenty four hour professional nursing care or removal to an institution Under any other circumstance he is wise if he disclaims responsibility and refuses to participate in the handling of the situation unless fortified by specialist consultation with explicit direction

Partial Suicide— Partial suicide is attempted by the demand of the patient for multiple surgical procedures The tonsils and adenoids are removed, the appendix and gallbladder are sacrificed the female genitalia are removed piecemeal or at one swoop In each instance the indications seem questionable and the surgical failure is described with a curious mixture of pride and dismay *The astute practitioner is wary of* the veteran of many surgical amphitheatres particularly if there are evidences of personality and conduct disorders In the presence of at least one relative the practitioner counsels reference to a trained psychiatrist

Management of Personality Disorders—The practitioner successfully deals with most conduct disorders though there is no method by which he may alter the personality pattern In general his management consists in an exploration of the troublesome situation correction of misinformation and in reeducation and adjustment of the involved individual

Exploration of the disturbing situation often reveals that the accused individual is not alone at fault More often than not particularly in childhood disturbance the disorder rests with misdirected zeal a neurotic tendency or misinformation on the part of the parents Crying spells and terror dreams with bed wetting often result from horror stories in the fairy tales or heard on the radio Lying and stealing are devices used to escape inordinately severe punishment for minor acts of naughtiness The child of a matriarch may respond by complete loss of initiative or an arrogant hostility

Many of the adolescent difficulties and maladjustments of adult life stem from the large mass of *absolutely authentic* (*mis*)*information* with which each individual is bombarded from all sides The majority of misconceptions deal with sexuality It is a source of recurrent amazement to note how much good can be accomplished by a straightforward explanation of sexual hygiene In households where it seems impossible to accomplish a satisfactory adjustment it may be necessary to send a child to boarding school or a summer camp The aged are often happier and better cared for in institutions devoted to their problems

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CHAPTER 65

PSYCHIATRY THE PSYCHOSES

THE psychoses are disorders of total personality Their differentiation from the neuroses has been given previous consideration (Table 86 p 1313) The psychoses are subdivided arbitrarily into idiopathic and symptomatic varieties

SCHIZOPHRENIA (DEMENTIA PRAECOX)

The schizophrenic reaction is a psychotic disturbance characterized by a cleavage or splitting between ideational contents and affect Closer to the core of the schizophrenic process however is the disturbance in the accurate relationship to external reality Since the disturbance is encountered most often in the 'teen age groups and the early twenties it has also come to be known as dementia praecox or the nervous breakdown of the young

Schizophrenic reactions constitute a grave and frequent type of mental disease It is estimated that 40 000 patients annually develop dementia praecox in the United States The vast majority of these require institutionalization and since the course is usually progressive without striking therapeutic benefits the span of occupancy averages one to two decades to the detriment of all concerned

The Schizoid Personality—Schizophrenic reactions usually but not invariably develop in individuals of the so called schizoid personality Since the manifestations of the latter are usually first observed by the practitioner at a time when treatment offers the best possibility for benefit it is important to stress these traits as harbingers of later danger

Alarming personality traits which arouse suspicion include the *brooding post adolescent* for whom no one is good enough and whom nothing pleases the *antisocial person* who is overcritical of everyone and everything, who finds satisfaction only in himself and in his fantasy life and

who hides behind superior attitudes the *persistent masturbator* especially the one who is worried about his habit fights unsuccessfully to control it and has great feelings of guilt relative to it the *intense bookworm* who is so preoccupied with philosophic speculations and the quest for the absolute that he misses the direct satisfaction obtained from relatively simple outlets such as interpersonal relationship the *overreligious* especially those preoccupied with mystical elements and religion and those who personalize experiences the *stubborn unpersuadable person* who is indifferent to the emotional needs of others and is given to frequent displays of temper the *odd individual* whose behavior is marked by strange quirks who is unsocial and seemingly oblivious to the ordinary demands of the community the *tense shut in* who can open up to no one and who is unable to share painful situations or entrust difficult decisions to those whom he seemingly loves and trusts

TABLE 87—DIFFERENTIATION OF IDIOPATHIC AND SYMPTOMATIC PSYCHOSES

	Idiopathic Psychosis	Symptomatic Psychosis
History	Of personality disturbances (p 1359)	Of definite trauma infection or intoxication
General physical status	Negative	Positive findings in syphilis and vascular diseases
Neurologic physical examination	Negative	Positive findings particularly in syphilis following encephalitis or meningitis and with neoplasm
Cerebrospinal fluid	Negative	Positive findings in syphilis encephalitis and meningitis
Other laboratory findings	Negative	Positive findings in syphilis metabolic disturbances such as hyperthyroidism and diabetes mellitus renal or hepatic insufficiency and hyperchromic anemia

In brief the practitioner is wary of young people who are acutely out of harmony with themselves their families and their community and for whom the usual adjustments in the art of living have been too painful and too difficult

Clinical Manifestations—The schizophrenic reaction is usually simple paranoid catatonic or hebephrenic

Simple Schizophrenic Reaction—The simple schizophrenic reaction consists of a gradual withdrawal from reality The patient becomes increasingly dull and apathetic he shows no interest in the world at large or in his smaller immediate world and he lacks ambition and initiative Despite apathy and indifference the schizophrenic often becomes irritable and moody for no consistent conscious reason Mental and intellectual deterioration slowly develops Hallucinatory experiences and delusions are rare *Neglected individuals who suffer from this reaction sink into prostitution* vagrancy and hoboism

CHAPTER 65

PSYCHIATRY THE PSYCHOSES

Schizophrenia (Dementia Praecox)
Manic Depressive Insanity
Involutional Psychoses
Paranoia and Paranoid Reaction Types
Traumatic Psychoses
The Febrile Psychoses
Syphilitic Psychoses
Encephalitic Psychoses
Psychoses Following Tuberculous and Other Forms of Meningitis
Psychoses in Neoplastic Disease
Presenile Psychoses (Pick Alzheimer)
Senile Psychoses
Post apoplectic Psychoses
Psychoses Associated with Endogenous Metabolic Conditions
Psychoses Associated with Exogenous Metabolic Conditions
Psychoses Occurring with Miscellaneous Disorders

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Alarming personality traits which arouse suspicion include the *brooding post adolescent* for whom no one is good enough and whom nothing pleases the *antisocial person* who is overcritical of everyone and everything, who finds satisfaction only in himself and in his fantasy life and

Hebephrenia —The hebephrenic schizophrenic reaction type is the least clearly defined It is characterized by silly behavior grotesque mannerisms incoherence of thought inadequate and seemingly unmotivated emotional responses laughter and crying

Etiology and Pathogenesis —The etiology and pathogenesis of the schizophrenic reaction types remain a mystery The psychoanalytically trained psychiatrists emphasize the role of emotional conflict Others deprecate these factors and stress constitutional hereditary and biological factors If there is a hereditary basis for dementia praecox it is yet to be proved Whereas schizophrenia occurs more frequently in certain families it may arise in the midst of perfectly good inheritance

There is no known pathology of schizophrenia Questionable changes are described in the brain and other organs especially the endocrine glands but these probably are secondary and are characterized by being highly inconsistent

Diagnosis —The diagnosis of schizophrenia depends upon the personality determinants The physical examination reveals no abnormalities and

TABLE 58 —RESULTS OF ELECTRIC CONVULSIVE THERAPY IN C SE OF SCHIZOPHRENIA

	Total	Recovered and Much Improved	Improved	Unimproved
Less than 6 months	60	41 (68 3 per cent)	13 (21 7 per cent)	6 (10 per cent)
6 months to 2 years	8[illegible]	34 (41 5 per cent)	24 (9 3 per cent)	24 (9 3 per cent)
More than 2 years	87	8 (9 2 per cent)	36 (41 4 per cent)	43 (49 4 per cent)
Old cases with previous remissions	46	26 (56 5 per cent)	10 (21 7 per cent)	10 (21 per cent)
Total	275	109	83	8[illegible]

Kalinowsky Bull N Y Acad of Med Vol 20 No 9 September 1944

there is no assistance other than of a negative quality to be obtained from the laboratory

There should be no difficulty in differentiating schizophrenia from the neuroses since the latter as has been previously related are partial personality disorders (p 1315) The greatest problem arises in determining where the schizoid personality ends and the psychotic schizophrenic reaction begins This qualitative decision is best left to the consulting psychiatrist who in point of fact may require weeks or months of observation before a definite decision is forthcoming

Prognosis —There should be no tendency to underrate the gravity of the prognosis in schizophrenia Nevertheless the recent introduction of the various forms of shock therapy have somewhat relieved the previous outlook of utter hopelessness In general patients who have had a relatively acute onset have a better response to treatment Older patients and those who have had the disease for any length of time deteriorate emotionally and intellectually They run a chronic and subchronic course and form a huge case load in the mental disease hospitals

Paranoia—The paranoid schizophrenic reaction type is characterized by active delusions of a persecutory or grandiose nature with well marked ideas of reference (p 1297) Auditory and visual hallucinations are common (p 1293) The patient's life rapidly becomes disorganized He is quarrelsome and suspicious He gives up work and may refuse to eat because of the fear that the food is being poisoned He may commit acts of violence and destruction in response to hallucinatory experiences Hence the paranoid schizophrenic comes in conflict with law and enters the category of the 'criminally insane

Catatonia—In the catatonic schizophrenic reaction types there are fluctuating episodes of depression excitement and stupor These may be preceded by other schizophrenic manifestations but often are fairly acute

DIFFERENTIAL DIAGNOSIS OF

Idiopathic Psychoses

The differential diagnosis of the idiopathic psycho es is dependent upon clinical fe t res since neither physical nor laboratory findings are pathognomic

VARIETIES	DIAGNOSTIC FEATURES
Schizophrenia	Patients between 13 and 25 years old History of eccentricity in adole cence Manifestations may consist of withdrawal from reality , paranoia or catatonia (p 1364)
Manic Depressive Insanity	Usually occurs after 25th year History of swings in mood from gayness to sadness Manife tations may feature phases of expansion or depression with remissions (p 1368)
Involutional Psychoses	After climacteric in females more frequently than in males History of prior maladjustment Manifestations often related to psychosexual inadequacy (p 1372)
Paranoia	Schizoid phenomena in older patients Manifestations usually delusional or persecutory (p 1373)

in their onset In a stuporous phase the patient sits or lies about quite cut off from the world of reality There may be negativism or waxy flexibility (p 1310) in which bizarre positions are maintained without change for hours Lesser degrees of stupor are characterized by great indifference stereotype of action and words (p 1309) echopraxia and echolalia (p 1309) Stuporous patients often hallucinate and have delusions of persecution or grandeur (p 1296)

Extreme excitation may alternate with the stupor Under these conditions the language and behavior are transformed into intense violence and homicide and suicide may occur Hallucinations or delusions are present and the excitation lasts for several days until the stupor again supervenes

may precede a *fully* developed phase of excitation *Contrariwise the hy* pomanic may develop irritability and a process of slowing down as if suddenly he were compelled to leave a concrete road and continue his course in heavy sand or mud

Phase of Expansion—Evidences of the illness appear characteristically in the spheres of the emotional and mental life the mood stream of thought and psychomotor activity In the *expansive phase* the mood is one of elation supreme self confidence and increasing assertiveness The patient becomes restless and easily distracted The speech at first is vol uble and high pitched Later the content has no conscious direction and is determined by the mere sound (*clang association*) There is true flight of thought with a rapid shifting from idea to idea Stimuli pour from within and without and no one idea is fully developed or completed The patient loses all the ordinary self regulation of behavior and becomes obscene and exhibitionalistic with uninhibited eroticism and excessive drinking particularly dipsomania The phase may terminate in a state of delirious mania with extreme violence uncontrollable excitement and a tendency to destruction

Phase of Depression—In the depressed phase the individual seems ex cessively timid hyperconscientious prudish shy and self deprecatory He is given to worry about petty details especially those having to do with money food cleanliness and toilet hygiene The complaints include easy *fatigability a sense of weakness depression the blues headache fre* quently in the back of the neck gastro-intestinal upsets with loss of appetite distaste for food and bad taste in the mouth restlessness and unsatisfactory sleep in which the patient awakens more exhausted than when I went to sleep impotence and frigidity There is a falling off in the accustomed standards of adequacy and efficiency The sufferer finds himself unable to make decisions in business or personal life he is beset by fears which he realizes are not entirely valid he seems disinclined to pursue the usual social activities and prefers to be left alone he finds *social intercourse a strain and conversation difficult Finally he decides* that he needs a rest or a change he finds that he ought to leave his job get out of a rut or get away from it all

Gradually or sometimes suddenly the depression deepens and the pa tient feels hopeless and despondent There is a sense of impending disaster which makes the future seem black and the prospect unrelieved By this time the patient looks depressed and miserable The expression is fixed and tight lipped furrows deepen the body seems stooped it is difficult to move and actions become increasingly slow Left to himself the patient *may sit for hours without moving Thinking is slow and retarded speech* is monosyllabic and there may be total mutism Decisions are difficult to make and painful The slightest detail looms gigantic and is weighted with great emotion Ideas of remorse and self depreciation appear Ancient trivial mistakes are revived and magnified Self accusations are hurled guilt mounts without control delusions of persecution or poverty and hy pochondriacal thoughts are frequent

Often the unwillingness to eat produces great loss of weight and a lowering of the basal metabolic rate There may be severe constipation and amenorrhea phenomena which are secondary *and not to be inter*

Treatment—The schizophrenic is institutionalized for adequate therapy referable to his own problems and for the protection of the immediate family the physician and the community

The modern treatment of schizophrenia requires some form of *shock therapy* through the use of insulin (p 1330) the medullary convulsants or by electrical methods (p 1330) These technics are the province of the specialist and are combined with psychotherapy in the attempt to arrest the process and maintain relatively normal interludes A judicious interweaving of shock and psychoanalytic therapy, in early schizophrenic reactions, accomplishes more than either modality without the other

A surgical approach to the treatment of schizophrenia is the technic of prefrontal lobotomy involving a severance of fiber tracts through a trephine opening An occasional brilliant result is achieved warranting consideration of the procedure in expert hands

MANIC-DEPRESSIVE INSANITY

The manic depressive psychosis is a highly important and common reaction type It is observed very frequently in general practice and is characterized by phasic episodes of excitement and depression Aside from the more striking examples which constitute 10 to 50 per cent of patients admitted to mental hospitals *masked forms* of the depressive phase are seen with disturbing frequency by the family practitioner

Prepsychotic Personality Types—It is important for the practitioner to recognize the prepsychotic personality types of the manic depressive reaction He has daily experience with personalities given to wide and unpredictable swings in mood from gayness to sadness These seem unmotivated by anything in reality and apparently arise without obvious cause

Depressions—Most frequently the practitioner observes episodes of depression which recur at irregular intervals and deepen during some part of the day They may or may not be interspersed with periods of elation In these phases common complaints include apathy fatigability lack of pep' disinterestedness the blues loss of ambition and initiative inability to do intellectual work and loss of potency or appetite Everything seems to be just too much and nothing seems worthwhile

Elations—Less often the practitioner recognizes the hypermanic personality characterized by excessive energy constant bustling limitless pep absence of fatigability a superficial interest in a myriad of things readiness for fresh undertakings in business or pleasure and avidity for new experiences thrills excitement or sexual contact

For the most part prepsychotic personality types present no important clinical finding It is only when the degree of involvement becomes sufficient to interfere with the normal activities of life that the practitioner is consulted At this time prophylactic psychotherapy may be launched particularly in a depression with promise of preventing the development of the fullblown psychosis

Clinical Manifestations of Manic Depressive Psychosis—Most often the manic depressive psychosis arises in the patient with the prepsychotic personality type The transition may be as abrupt as the falling of a curtain or it may be imperceptible A brief almost unnoted period of depression

may precede a fully developed phase of excitation Contrariwise the hypomanic may develop irritability and a process of slowing down as if suddenly he were compelled to leave a concrete road and continue his course in heavy sand or mud

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preted as primary or etiologic in their significance In the advanced stages the patient goes into stupor, and psychomotor activity speech and cerebration are suspended

Etiology and Pathogenesis —The etiology of the manic depressive psychosis is unknown *Psychogenic elements* unquestionably play a role The psychoanalytically trained psychiatrists lay emphasis on these factors but most of the eclectic school of neuropsychiatrists regard the disturbance as *metabolic* or *toxic* in origin There seems no doubt as to the existence of a *hereditary factor* since the manic depressive psychoses are twenty five times as frequently observed among the siblings of previous sufferers as in the general population The manic depressive reaction occurs twice as commonly in women When it occurs at the menopausal time it is regarded as an *involutional psychosis* (1372)

Nervous Breakdown —Occasionally there appears an exciting factor such as the loss of a job disappointment in a love affair or a prolonged illness In most of these examples of so called 'nervous breakdown' it is more likely that the apparently causative agent is a precipitant and merely coincided with or provoked the deeper underlying disturbance

In no instance are there any demonstrable pathologic or significant laboratory findings

Diagnosis —A complete diagnostic survey is conducted before subscribing to the diagnosis of manic depressive psychosis The agitated phase may be mistaken for *hyperthyroidism* (p 1197), and the depressed phase is simulated by *hypothyroidism* (p 1191) the early phases of subacute or chronic *infection* particularly *tuberculosis* the premonitory manifestations of chronic and debilitating diseases such as *carcinomatosis pernicious anemia* the *leukemias infectious mononucleosis* and *brucellosis*

In addition to a record of the temperature curve and the completion of a meticulous physical examination the laboratory investigation includes a full blood count urinalysis estimation of the basal metabolic rate radiography of the chest and serologic reactions for syphilis and brucellosis

Course and Prognosis —In contrast to the ominous outlook in the schizophrenic reaction the prognosis in the manic depressive episode holds considerable promise Proportionately only a small percentage of the afflicted develop to the stage where they require institutionalization and treatment often offers a bright if temporary prospect

Treatment —In the treatment of the earlier mild phases of excitement and depression the general practitioner functions quite successfully He comes to the situation with certain distinct advantages since in most instances the patient knows him and as far as his illness permits has confidence in him

Nontechnical Psychotherapy —Especially in the case of a patient with hypochondriacal anxiety a thorough going and complete *physical examination* is an essential first step It must be sufficiently complete to justify the physician s contention that there is nothing organically wrong It should not be too fussy or specialized as to suggest to the already suspicious patient that there is hidden or subtle disease The physician must not expect overmuch from his assurance knowing that his psychotherapeutic effort is opposed by the patient s need or will to be ill' He must be prepared patiently to hear out the patient he must be ready to sit

through and be interested in the mass of detail which seems to be so pressing and important to the patient Not infrequently this material contains the clue to an irritant or conflict which can be judiciously dealt with on a superficial basis

The depressed patient s need for reassurance must not be answered by attempts to minimize the complaints or the conveyance of the idea that he is really not as sick as he thinks The patient must not get the notion that a complaint which is very real and disturbing to him is trivial or fictitious to his physician As in any psychotherapeutic relationship the realness of the patient s complaints must be a premise Never should the physician imply that the difficulty is imaginary For indeed it never is! The difference between unreal and imaginary and psychogenic or emotionally induced must be meaningful to the physician if he is successfully to communicate that distinction to his patient

Drug Therapy—Many depressed patients receive a considerable lift from *amphetamine sulfate* given in doses of 2.5 to 10 mg each day The mildly agitated are sedated with *barbiturates* during the day and are given a *hypnotic* at bedtime

Other recommended preparations which probably have no specific value other than through the power of suggestion include *strychnine caffeine neurophosphate thiamine chloride* and others of the vitamins *androgen estrogen adrenal cortical extract* and *epinephrine* In order to encourage the return of the patient for psychotherapy any one of these substances may be injected by hypodermic The physician who highly regards the specificity of the injected product is a better psychotherapist than the skeptic

Hydrotherapy—The ambulatory treatment of the depressed patient is often aided by home *hydrotherapy* (p 3792) The advances in modern plumbing provide for a warm relaxation tub bath a cold shower or shock treatment provided by buckets of cold water

Change in Environment—Every effort should be made to keep the patient in his environment despite his desire to escape and seek other surroundings or another job With continuation of the symptoms and persistent urgings on the part of the patient it may be necessary to yield and agree to a holiday such as a fishing or a hunting trip or a visit to the mountains or the seashore or to relatives or friends A change of job however should not be countenanced until the vacation has been tried

In contrast to the depressed patient the agitated individual must be prevented from entering upon flamboyant enterprises which may involve the patient his family and friends in financial havoc

Formal Psychotherapy—Formal psychotherapy in an ambulatory fashion is advisable for patients who are not sufficiently ill to require institutionalization and yet who fail to respond to the practitioner s nontechnical endeavors Those who advocate psychoanalysis maintain that their efforts often control the fluctuation in the manic depression reaction and spare the patient from the more serious ravages of the disturbance

Institutionalization—Institutionalization is required for the depressed patient when he becomes suicidal or can no longer care for himself In the agitated phases hospitalization is advisable to prevent difficulties resulting from excesses in drinking sexual desire financial investments or gambling

preted as primary or etiologic in their significance In the advanced stages the patient goes into stupor and psychomotor activity speech and cerebration are suspended

Etiology and Pathogenesis—The etiology of the manic depressive psychosis is unknown *Psychogenic elements* unquestionably play a role The psychoanalytically trained psychiatrists lay emphasis on these factors but most of the eclectic school of neuropsychiatrists regard the disturbance as *metabolic* or *toxic* in origin There seems no doubt as to the existence of a *hereditary factor* since the manic depressive psychoses are twenty five times as frequently observed among the siblings of previous sufferers as in the general population The manic depressive reaction occurs twice as commonly in women When it occurs at the menopausal time it is regarded as an *involutional psychosis* (1372)

Nervous Breakdown —Occasionally there appears an exciting factor such as the loss of a job disappointment in a love affair or a prolonged illness In most of these examples of so called nervous breakdown it is more likely that the apparently causative agent is a precipitant and merely coincided with or provoked the deeper underlying disturbance

In no instance are there any demonstrable pathologic or significant laboratory findings

Diagnosis—A complete diagnostic survey is conducted before subscribing to the diagnosis of manic depressive psychosis The agitated phase may be mistaken for *hyperthyrodism* (p 1197), and the depressed phase is simulated by *hypothyroidism* (p 1191) the early phases of subacute or chronic *infection* particularly *tuberculosis* the premonitory manifestations of chronic and debilitating diseases such as *carcinomatosis pernicious anemia* the *leukemias infectious mononucleosis* and *brucellosis*

In addition to a record of the temperature curve and the completion of a meticulous physical examination the laboratory investigation includes a full blood count urinalysis estimation of the basal metabolic rate radiography of the chest and serologic reactions for syphilis and brucellosis

Course and Prognosis—In contrast to the ominous outlook in the schizophrenic reaction the prognosis in the manic depressive episode holds considerable promise Proportionately only a small percentage of the afflicted develop to the stage where they require institutionalization and treatment often offers a bright if temporary, prospect

Treatment—In the treatment of the earlier mild phases of excitement and depression the general practitioner functions quite successfully He comes to the situation with certain distinct advantages since in most instances the patient knows him and as far as his illness permits has confidence in him

Nontechnical Psychotherapy—Especially in the case of a patient with hypochondriacal anxiety a thorough going and complete *physical examination* is an essential first step It must be sufficiently complete to justify the physician s contention that there is nothing organically wrong It should not be too fussy or specialized as to suggest to the already suspicious patient that there is hidden or subtle disease The physician must *not expect* overmuch from his assurance knowing that his psychotherapeutic effort is opposed by the patient s need or will to be ill He must be prepared patiently to hear out the patient he must be ready to sit

forgivable sin and cloaked with a profound sense of guilt The personality quirks and eccentricities become accentuated until insight is so far lost that the mental symptoms dominate all thinking and activity

Treatment —The minor personality disturbances of the involutional period are managed in the manner of the *male* and *female climacteric* (p 2523) Fully developed involutional psychoses require *institutionalization* Shock therapy (p 1330) may be of significant value in association with adjuvant *psychotherapy and injections of androgen or estrogen*

PARANOIA AND PARANOID REACTION TYPES

Psychiatrists dispute the nature of true paranoia in its relation to the paranoid reactions of schizophrenia The present discussion avoids *these issues and is content with descriptive detail*

Etiology —The etiology of paranoia is unknown Psychogenic elements probably play a large if not exclusive determining role The prepsychotic personality varies but frequently the illness is encountered in people who have always been more or less suspicious or mistrustful who have been stubborn and resentful of criticism and have found refuge in sulking or in fantasy life In these patients the psychosis seems but a continuation and *acceleration of a customary pattern for dealing with conflicts* especially unconscious ones The Freudian explanation for paranoia stresses the role of unconscious homosexuality which is *generally accepted* as an important element in the production of illness

Clinical Manifestations —The outstanding characteristics of paranoia are the presence of central *delusional themes* most frequently *persecutory* or *grandiose* The *idee fixe* is systematized and unchangeable It develops insidiously unrelated to toxic or organic causes and unassociated with an emotional or intellectual dilapidation of the general personality

The distrust and suspiciousness of the prepsychotic state *increases* Everything is interpreted by the patient as relating to him (ideas of reference) he finds hostility everywhere the inner tensions increase with ever increasing vehemence and rigidity and his conflicts drive him to defend his delusional patterns Even past events are reinterpreted to fit the delusional need and the patient finds in them evidence of which he had not himself been aware when they were occurring (retrospective falsification)

At first glance the arguments of the paranoiac are beguilingly con*vincing An attentive hearing however soon reveals weaknesses Although* granting the original false premise they proceed with a certain logic and plausibility The patient appears too tense too anxious too sullen and uncommunicative when pressed as though in an unconscious sense he was aware of the frailty of his thesis The delusions however are never long subjected to reason and no explanation other than the delusional one is even tenable

In certain types of the paranoid reactions the delusional pattern is accompanied by varying degrees of personality disintegration approaching the status of a paranoid *schizophrenia Paranoids confronted with jealousy* and hostility not infrequently resort to alcohol See p 3847

Prognosis —Though the paranoid may continue to live a fairly adequate life outside of a hospital recovery never occurs The mild varieties include

Shock Therapy—The depressed phase of manic depressive states responds brilliantly to *shock therapy* using insulin the medullary convulsants or electrically induced seizures Perhaps the best results are obtained by the combination of shock therapy during the height of the psychosis with supportive psychotherapy during the free phases

INVOLUTIONAL PSYCHOSES

It is difficult to estimate the role of endocrine dislocations associated with the climacteric in the causation of involutional psychoses Many psychiatrists believe that separate classification is unwarranted and regard the involutional changes as mere additional strains in the total adjustment of the personality Others interpret the metabolic and hormonal changes as of prime importance in pathogenesis

Clinical Manifestations—A review of the personality of most patients discloses frank signs of prior maladjustments with timid inhibited worr

TABLE 89—RESULTS OF ELECTRIC CONVULSIVE THERAPY IN 800 CASES OF THE AFFECTIVE DISORDERS

	Total	Recovered and Much Improved	Improved	Unimproved
Manic depressive (depressed)	60	52 (86 6 per cent)	4 (6 7 per cent)	4 (6 7 per cent)
Manic depressive (manic)	32	27 (84 4 per cent)	3 (9 4 per cent)	2 (6.2 per cent)
Involutional Melancholia	76	66 (86 9 per cent)	8 (10.5 per cent)	2 (2 6 per cent)
Involutional Paranoid	32	14 (43 7 per cent)	8 (25 per cent)	10 (31.3 per cent)
Total	200	159	23	18

Kalinowsky Bull N Y Acad of Med Vol 20 No 9 September 1944

some frugal overconscientious and masochistic tendencies, compulsive drives rigid attitudes and a deep sense of insecurity The associated climacteric changes superimpose additional psychological stresses which may contribute substantially to the full development of the psychosis

The menopause does have a profound emotional impact on an unhappy insecure woman and failing potency disturbs the man who had never been too secure in his psychosexual adjustments False teachings have much to do with many conflicts especially in women who are brought up to look with dread toward the oncoming change of life and expect the worst who believe the menopause demands celibacy and drive their husbands to continence or philandering Industry with few jobs for men beyond the age of forty adds to the hollowness and insecurity of those who have not been successful in their careers

Characteristic of the involutional psychoses are delusional ideas commonly of a persecutory nature feelings of guilt and self deprecatory ideas The most trifling indiscretion in youth is resurrected interpreted as an un

Metabolic	With avitaminoses diabetes mellitus (p 1246) episodes of hypoglycemia (p 734) renal insufficiency and azotemia (p 2276) hepatic insufficiency and cholemia (p 1953) hypothyroidism and hyperthyroidism (p 1197) *and acidosis (p 721) Get urinalysis blood* chemistry and B M. R. Try therapeutic tests with soluble vitamins iodides and glandular extracts (p 1149)
Pharmacologic	Following therapeutic administration of general anesthetics sedatives hypnotics cocaine opiates atropine scopolamine bromides arsenicals, mercurials and sulfonamides
Toxicologic	In drug addicts particularly with marihuana With lead, mercury manganese and carbon disulfide poisonings in industry In carbon monoxide poisoning
With Multiple Sclerosis	Mental changes in conjunction with temporal *pallor of optic disc and diffuse motor and sensory manifestations.*
With Hyperchromic Anemia	Mood changes in association with abnormal *ities of the hemogram (p 1077)*
Epileptic	Delusions of persecution and mental deterioration most often precipitated by bromide intoxication

in their roster many of the cranks the habitual litigants the chronic writers of letters to the authorities and press and the alcoholics When the paranoid reaction is a secondary feature as in paranoid schizophrenia *occasional spontaneous but partial remissions may occur*

Treatment—Ambulatory treatment is highly ineffectual *Hospitalization* is required from the point of view of the protection of the community and especially those against whom the delusions are directed shock therapy and prefrontal lobotomy are worthy of consideration if medicolegal barriers can be overcome

TRAUMATIC PSYCHOSES

Traumatic psychoses constitute reactions that occur after injury The trauma may be applied directly to the head or it may be inflicted on other portions of the body Under any circumstance the history usually reveals a *preexistent personality conflict* which under the influence of the traumatic experience becomes activated

Trauma also precipitates clear psychotic reactions The first obvious manifestation of a schizophrenic reaction a manic-depressive psychosis or a general paresis may seem related to injury a fact which is often a subject for violent medicolegal argumentation

Psychoses of Combat—The violence of modern war has added immeasurably to the incidence and importance of traumatic psychoses To the *shell shock* of World War I have now been added the mental disturbances that result from prolonged submersion in submarines from the daily hazards in the merchant marine with the ever present dangers of bombing and torpedoing and from the inhuman conditions in aviation where men travel at phenomenal speeds in and below the stratosphere and where the

DIFFERENTIAL DIAGNOSIS OF

Symptomatic Psychoses

Symptomatic psychoses are differentiated from idiopathic varieties by definite evidence of their cause in the history physical and neurologic status examinations cerebrospinal fluid findings or the electroencephalogram

TYPE	DIAGNOSTIC FEATURES
Traumatic	History of injury Clinical manifestations dependent upon prior per onality pattern.
Febrile	Usually associated with acute hyperpyrexia or prolonged and exhausting infection. Acute varieties may become manifest through mania or coma Protracted type often as ociated with avitaminoses
General Paresis	Syphilitic encephalo meningitis Wassermann tests positive in blood and spinal fluid. Com plete decolorization in first five tubes of colloidal gold reaction Associated changes in pupils and deep reflexes Favorable response to specific therapy (p 1379)
Po t encephalitic	Hi tory of infection with cranial nerve palsies and spinal fluid pleocytosis Manifestations usually involve total per onality and sleep pattern
Post meningitic	History of fever with nuchal rigidity headache and generalized eruption Tubercle bacilli or pathogenic cocci isolated from cerebrospinal fluid (p 3737)
Choreic	Behavior di orders associated with choreiform movements and evidences of carditis Sedi mentation time usually increa ed (p 190)
Neoplastic	Various psychotic reactions may accompany brain tumor Make complete survey includ ing neurologic status (p 3584) fundus ex amination (p 1545) spinal fluid studies (p 3737) ventriculogram (p 3783) electroen cephalogram (p 1406) and exploratory crani otomy if necessary
Pre senile Psycho es	Occurs between ages of 40 and 60 Associated with other evidences of arteriosclerosis par ticularly retinal changes hypertension and nephropathy
Senile Psychoses	After 65 Accentuation of personality defects Usually associated with weight loss and mani festations of generalized arteriosclerosis (p 1382)
Post apoplectic	History of cerebral accident usually hemi plegia (p 1439)
Cardiovascular	In association with backward failure (p 941) pericardial effusion (p 1008) acute and chronic pericarditis (p 1010) and cardiac tamponade (p 872)

Metabolic	With avitaminoses diabetes mellitus (p 1246) episodes of hypoglycemia (p 734) renal insufficiency and azotemia (p 2276) hepatic insufficiency and cholemia (p 1953) hypothyroidism and hyperthyroidism (p 1197) and acidosis (p 721) Get urinalysis blood chemistry and B M R. Try therapeutic te ts with soluble vitamins iodides and glandular extracts (p 1149)
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human economy is subjected to the pitiless strain of going into and out of power dives"

Clinical Manifestations—The clinical manifestations of the traumatic psychoses depend in great part upon the personality pattern that preceded the traumatic experience There may be excitation and mania or depression and coma

The psychosis is often followed by late personality disturbances after the patient is considered well He becomes irritable nervous fearful apprehensive sleepless depressed and moody He suffers from headache fatigue lack of concentration or the will to work He smokes excessively, and soon begins to take too much coffee benzedrine alcohol and barbiturate and too little food

The post traumatic period is also followed by moral and social deterioration Disturbances in sexuality are observed as well as a general mental impairment A familiar illustration of the latter is the syndrome of *'punch drunk* seen in pugilists

Diagnosis—The diagnosis of traumatic psychosis is the province of the *specialist* especially in compensation and liability suits

Treatment—The treatment of the traumatic psychosis is *specialist province* under *institutional* auspices It involves a complex program including psychotherapy reeducation and revaluation hypnagogic reverie physiotherapy hydrotherapy occupational treatment and rehabilitation features Since the prognosis varies between complete recovery and more or less invalidism the efficacy of any therapeutic modality is difficult of evaluation The practitioner who assumes responsibility for his patient is subjected to a thankless criticism and the nuisance and embarrassment of the litigious features

FEBRILE PSYCHOSES

Pyrexia and hyperpyrexia often are associated with psychiatric derangements Adult patients with systemic infections become drowsy and comatose disoriented restless or delirious To add to diagnostic difficulties infants and children are prone to develop convulsions See p 2780

Clinical Manifestations—Most commonly the febrile psychosis occurs *at the onset* of an acute episode of hyperpyrexia Next in frequency the psychosis is encountered in the *long fevers* when metabolic derangement cumulative drug poisoning exhaustion and vitamin deficiencies are added to the disturbances of the febrile process The febrile states commonly associated with psychoses include pneumococcus pneumonitis typhoid fever and prolonged sepsis usually of streptococcal origin

The occurrence of psychiatric symptoms in a course of a febrile disease suggests possible infectious invasion of brain tissue When convulsions are added to the other disturbances a *lumbar puncture* is indicated for diagnostic and therapeutic purposes

Prognosis—The febrile psychosis that occurs early in a disease does not add appreciably to the gravity of the prognosis The late psychosis is an evil prognostic omen partially because it indicates a severe reaction and partially because it interferes with nursing care The febrile psychosis rarely produces permanent disturbances When protracted derangements occur there is a strong likelihood that a personality disorder preceded the

infection and that the psychosis was precipitated rather than was caused by the acute illness

Treatment —The febrile psychoses are treated with a minimal use of drugs Constant observation by experienced nurses is mandatory since patients may cause irreparable damage by leaving bed tearing off dress ings and intravenous infusion sets and by pulling out catheters and drain age tubes Serious injuries of course result when patients fall out of bed or jump from windows

Such disturbances are best controlled by *hydrotherapy* Sponges the application of ice bags to the head and the calming influence of a con scientious and intelligent nurse often suffice for sedation As soon as pos sible a continuous *intravenous drip* is instituted and the administration of fluids with dextrose saline and the soluble vitamins often gives amaz ing and seemingly specific relief If these measures alone do not suffice for sedation *paraldehyde* in a dose of 1 to 4 cc is injected into the rubber tubing to produce an immediate and often reasonably prolonged sleep When the patient shows signs of renewed restlessness a further dose of paraldehyde is given

Should the paraldehyde fail to be effectual *soluble barbiturates* (p 3839) are employed The use of morphine and related substances is hazard ous since many delirious patients are actually suffering a cat reaction as the result of previously administered opiate See p 3658

SYPHILITIC PSYCHOSES

Syphilis of the encephalomeningeal structures most commonly produces the syndrome of *general paresis* Additionally however there are observed nonparetic syphilitic disturbances such as *taboparesis* and *interstitial vas cular meningeal* or *gummatous* changes in the brain tissues

GENERAL PARESIS

The symptoms of paresis usually follow the acquisition of the primary lesion by an interval that may be as brief as five years and as long as twenty five years The peak of incidence is between thirty five and forty five years of age and the frequency of the disease may be estimated from the fact that it constitutes 8 per cent of all first admissions to mental hos pitals This does not represent the total figure for the disease since early cases are increasingly being treated in general hospitals

It is likely that the spirochete invades the brain at the time of the secondary stage The spinal fluid findings then are positive What it is that produces the period of latency and what activates the disturbance at the present time remain unanswered See p 340

Pathology —The brain in general paresis is small and atrophic espe cially over the frontal lobe The dura is thickened edematous and firmly attached to the skull There may be a subdural hemorrhagic pachymenin gitis The leptomeninges are cloudy and the arachnoid is firmly attached to the pia External and internal hydrocephalus produce flattening of the gyri and widening of the sulci The dura is infiltrated with new connective tissue and the pia is invaded by lymphocytes and mast cells The adven titial spaces contain lymphocytes plasma and giant cells and to some extent the infiltrations extend free into the tissues Blood vessels are in

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patients treated later the recovery rate falls to 30 to 35 per cent but the progress of the disease is checked in another 30 per cent

Treatment—In the era that preceded the introduction of penicillin in the treatment of syphilis the management of general paresis was conducted with eminent satisfaction through the use of hyperthermia With the combination of the two effective modalities even greater promise of significant therapeutic accomplishment is imminent

Penicillin—Particularly in asymptomatic paresis and in early manifestations of the disorder main reliance may be placed on penicillin therapy given intensively over a brief period of time Re-treatment at stated intervals may be required until greater knowledge of this extraordinary substance has been amassed For an initial course no less than 10 000 000 units of penicillin may be given by introducing 150 000 units intramuscularly every three hours for a period of slightly less than ten days Until more definite long term information is available the practitioner must be guided by the efficacy of this probatory course If spinal fluid clearing is not satisfactory at the end of three months the course must be repeated using perhaps 16 000 000 units in the same period of time This can be accomplished by increasing each individual injection to 200 000 units given at three hour intervals for an eight day period Whether more can be accomplished by a continuous intravenous drip of even more massive unitage remains for the future to decide

Hyperthermia—In institutional practice the experienced expert utilizes *cabinet treatment* In localities where hypertherms are not available fever therapy is accomplished by *blanket wrapping intravenous injections of typhoid vaccine* or the production of a *therapeutic malaria* (p 509) The first two technics are more strictly within the realm of the practitioner who rarely has access to a supply of tested malarial blood

BLANKET WRAPPING—Blanket wrapping is accomplished without the necessity of purchasing supplies that are not accessible in the ordinary home The patient puts on long woolen socks and gets into a bed which has been protected with a large rubber sheet and a length of canvas He is wrapped with several thin blankets and a heavy woolen one Each limb the trunk and shoulders are individually encased and finally the canvas and rubber sheets are drawn around and held together so that only the face is exposed The oral temperatures and temporal artery pulses are recorded at thirty minute intervals Following each reading the patient takes a tumblerful of hot lemonade containing 0 6 per cent of sodium chloride and sugar to taste

With this technic a temperature of 102.2 F is usually reached in three or four hours and 104° F in five to six hours An attempt is made to maintain the hyperpyrexia between 104 and 105° F for six hours At the height of the fever restlessness may be controlled by the administration of morphine or liberal doses of a hypnotic

The blankets are loosened and the patient is cooled if the temperature rises above 105° the pulse rate exceeds 140 or the respiratory rate advances beyond 28 per minute Treatments are repeated at weekly intervals and their efficacy is judged by clinical and serological alterations

INTRAVENOUS TYPHOID VACCINE—The second available method for the production of hyperpyrexia under the conditions of clinical practice con

creased and new blood vessel formation is visible The ganglion cells are severely damaged, the microglia are tremendously proliferated and many rod and "gitter cells" are observed There is an increase in the iron content of the brain tissue the microglia and adventitial cells Spirochetes are found scattered throughout the brain largely in the gray matter

Clinical Manifestations —The clinical manifestations of general paresis include changes in emotional stability intellect and character Patients are frequently depressed with or independent of expansive moods Intellectual impairment is especially marked in the spheres that require attention concentration, mathematical calculation and memory for recent events Personality quirks become accentuated A common symptom is inability to remember what was said or done a few moments previously

Character changes are pathognomonic and occur early in the disease They may take the form of sudden streaks of unwarranted extravagance carelessness about personal hygiene in a previously fastidious individual unaccustomed sexual promiscuity excessive alcoholism or criminal behavior in a previously conformant personality

In addition to the psychogenic symptoms there may be physical manifestations such as excessive fatigability headache epileptic seizures speech difficulties incoordination of motor movement (so that the patient is unable to perform simple acts such as buttoning of clothes) unexplained elevation of temperature apoplectiform seizures and alterations in urination and defecation

Physical Examination —Physical examination of the paretic is usually characteristic There is loss of facial tone and a washing out of expression The pupillary reactions reveal the *Argyll Robertson changes* (p 1534) Often there are ocular and extra ocular muscle weaknesses tremors of the face and tongue incoordination and ataxia of the fingers speech defects such as slurring and elision of syllables and absent tendon reflexes Babinski reflexes are frequently found hypalgesia over nose and chest is common and there may be a malar flush in contrast to the pallor of the tabetic

Laboratory Data —The diagnosis of general paresis is clearly revealed by the laboratory data Usually the *blood tests* are positive as are the *spinal fluid reactions* Additionally the *colloidal gold reaction* reveals complete decolorization in the first five tubes The presence of the "paretic curve" establishes or refutes the diagnosis of general paresis beyond the shadow of doubt

Course and Prognosis —Untreated general paresis is a progressive disease Intellectual impairment progresses to a true dementia and expansive paranoid and somatic delusions develop along with disorientation in all spheres The expansive phase which is unduly emphasized is not as frequent or as characteristic as has been generally supposed The emotional reactions deepen with increasing depression elation or agitation The patient becomes unable to care for himself and is utterly confused disoriented and incapacitated Death usually occurs within two or three years after the first symptoms occur

Spontaneous remissions are experienced in a small number of cases and may last from a few months to five years With adequate treatment in a relatively early stage from 30 to 60 per cent of paretics are well enough to return to their usual occupations In the more advanced instances of

ENCEPHALITIC PSYCHOSES

Mental symptoms appear in acute and subacute lethargic encephalitis (p 449) In the actue stage the symptoms are those of any acute toxic infection with *delirium* predominating There is often interference with the *sleep pattern* and the manifestations may vary from intractable insomnia to prolonged somnolence Occasionally the clinical picture simulates that of the schizophrenic or the manic depressive reaction types

In the subacute or chronic phase especially in children there occur devastating *personality alterations* The child exhibits behavior disorders periods of depression and exaltation irritability loss of interest impulsive and uncontrolled antisocial behavior such as lying stealing running away from school and home or gross sexual offenses These children may become very obese or pitifully emaciated

The prognosis of post encephalitic psychoses is never good *Institutionalization is usually required owing to the difficulties that arise at home* and in school

PSYCHOSES FOLLOWING TUBERCULOUS AND OTHER FORMS OF MENINGITIS

With *meningeal involvement* whether due to the tubercle bacillus or the coccal forms (p 1462) there may be acute delirium periods of irritability and excitement and acute confusional states

In the insidious infections such as *chorea* initial symptoms may be *behavior disorders* such as excessive and unaccustomed sulking temper tantrums and disobedience (p 1359)

PSYCHOSES IN NEOPLASTIC DISEASE

In an alarming number of cases in which patients were admitted to psychiatric institutions for psychotic reactions autopsies have revealed unsuspected silent *brain tumors* (p 1419) *The phychotic patient is entitled* to a complete organic survey with spinal fluid examinations encephalograms ventriculograms electro encephalograms and if necessary an exploratory craniotomy

PRESENILE PSYCHOSES (PICK ALZHEIMER)

Presenile psychoses are relatively rare They occur characteristically between the ages of forty and sixty and run a rapid course ending in dementia and death

The *symptoms* of the psychosis are variable and depend upon the nature and *location of the vascular lesion and the personality type* that predisposes to the lesion In addition to alterations in the personality panel there may be focal manifestations such as aphasia apraxia or motor and sensory manifestations

The diagnosis of presenile psychosis is made by exclusion The syphilitic derangements are eliminated if the blood and spinal fluids are clear Inflammatory diseases produce cytologic manifestations in the spinal fluid The suspicion of brain tumor requires specialist consultation and exploratory craniotomy

The prognosis of the presenile psychoses is hopeless There is no form of treatment other than custodial care

sists of intravenous injections of typhoid vaccine The usual commercial typhoid vaccines contain 1 000 000,000 organisms to the cubic centimeter An initial injection should not exceed 10 000 000 organisms or 0 1 cc of a 1 10 saline dilution Subsequent doses are dependent upon the reaction to the trial amount

The injection of typhoid vaccine is given in the morning In the suit able reaction the patient has a chill within thirty minutes to two hours The temperature rises to 102° to 105° at which time a second injection using the same dose is administered An adequate response is one in which there is a minimal rise to 104° but a temperature of 107° should not be exceeded

Medicolegal Aspects —In a certain number of instances head trauma activates the symptoms of general paresis and medicolegal controversies arise Not infrequently the precipitating accident resulted from a *defect* in judgment that was itself an expression of the disease

JUVENILE PARESIS

General paresis occurs as the result of *congenital syphilis* The usual age of incidence is five to twenty five years and this complication is noted in perhaps 1 per cent of all afflicted

The general findings are similar to those in adult paresis except that many of the patients remain *feebleminded* and the course of the disease despite therapy, is almost always progressive As a rule the patient suc cumbs four to five years after the onset of symptoms with confusional states apoplectiform seizures or from intercurrent infection

TABOPARESIS

The coexistence of tabes dorsalis and general paresis is not infrequent the patient revealing the signs and symptoms of both afflictions *Treat ment* is directed along the lines of otherwise uncomplicated paresis (p 1379)

OTHER SYPHILITIC PSYCHOSES

In addition to the syndrome of general paresis the syphilitic brain suffers parenchymatous involvements due to vascular meningeal and gum matous changes Each of these alone or in combination may produce men tal changes of great variety These manifestations usually occur two to five years after the primary lesion

The clinical manifestations are varied They may consist of delirium with a defect in memory for recent events there may be alteration in mood toward depression or euphoria and with increased intracranial pres sure stupor may occur preceded by a state of dullness and apathy In the last circumstance the usual organic findings of increased intracranial pres sure are observed These include papilledema localized nerve palsy (par ticularly of the eye muscles) and a marked lymphocytic reaction in the spinal fluid

The prognosis of nonparetic syphilitic psychotic reactions is somewhat better than that of general paresis Specific *treatment* with or without hyperpyrexia often causes prompt disappearance of the disturbance (p 1379)

PSYCHOSES ASSOCIATED WITH ENDOGENOUS METABOLIC CONDITIONS

Psychoses may accompany a variety of endogenous metabolic conditions The febrile and post febrile psychoses have been previously mentioned Psychogenic derangements occur also in *hyperglycemia hypoglycemia acidosis azotemia hepatic insufficiency hypothyroidism hyperthyroidism* and *adrenal cortical deficiency* They are also seen in the *anoxemic states* that accompany *cardiac failure* and in *cardiac tamponade* due to a rapidly accumulating *pericardial effusion*

Psychoses accompany the *avitaminoses* particularly *pellagra* Almost 10 per cent of pellagrins show mental changes which may vary from deliria which are the most common to typical organic psychotic reactions Changes in mood especially depressions are common

The *treatment* of the psychoses is subservient to the correction of the more fundamental disturbance

PSYCHOSES ASSOCIATED WITH EXOGENOUS METABOLIC CONDITIONS

Psychoses develop from the administration of *drugs* and exposure to *chemicals* in industry Among the causative or provocative agencies are alcohol cocaine morphine barbiturates marihuana lead mercury arsenic sulfa drugs atabrine and bromides

BROMIDE INTOXICATION

The indiscriminate use of bromides available to the laity without prescription results in the superimposition of a drug psychosis on the structure of underlying mental or nervous diseases particularly epilepsy or alcoholism Along with the mental symptoms there may be a *bromide eruption (p 3339) the breath has a characteristic sweetish odor the speech* is thick and slurred there is general muscular incoordination ataxia tremors of the fingers and perioral muscles the pupils are widely dilated and react poorly to light and the deep reflexes are depressed or abolished

Treatment consists of stopping the drug forcing fluids and giving large quantities of sodium chloride if necessary by intravenous drip (p 3775) If the restlessness and excitement are intense paraldehyde is the most useful drug to use

PSYCHOSES DUE TO INGESTION OF HEAVY METALS

Psychotic reactions resulting from *lead* poisoning were once frequent At times plumbism produced a quite typical acute delirium and a slowly progressive intellectual dulling leading to a profound mental deterioration Less often there was an obscure mental reaction resembling neurasthenia with irritability restlessness easy fatigability feelings of depression and anxiousness

Similar clinical pictures result from poisoning with *mercury manganese* and *carbon disulfide* (p 747)

PSYCHOSES DUE TO GASES

Compared with the large number of *carbon monoxide poisonings* mental symptoms are relatively uncommon and usually occur only after pro

SENILE PSYCHOSES

Older people are expected to become increasingly egocentric They are apt to be fussy and irritable, and they expect and demand a great deal of attention They become less punctilious about their habits and their personal hygiene and they are forgetful and given to long winded reminiscences

Clinical Manifestations—The normal phenomena of the process of aging usually occur at the ages of from sixty to sixty five When the transition occurs at an early time or develops acutely, the condition is regarded as abnormal *in the quantitative sense The forgetfulness* develops into a loss of memory for most recent events the reminiscing becomes an endless repetition of ancient happenings detailed in the greatest degree, the egocentricity and irritability assume definite paranoid colorings and there are outbursts of unprovoked temper and childish sulking

The paranoid ideas lead to quarreling distrust of family and friends and reluctance to eat and compensatory protective reactions There is a hoarding of worthless objects such as matches pieces of string and left over food These patients wander from home they are improperly dressed they neglect toilet habits and personal cleanliness they become preoccupied *with sex and make indecent propositions and expose themselves* inordinately as though these practices compensated for lost sexual capacity Often they return to masturbation and puppy love confabulation is common and true delirium confusional states and delusional trends may occur

Treatment—As long as senile patients can be tolerated and cared for at home they are best taken care of under their normal surroundings Essentially, they need patience tact and understanding When the household becomes too greatly disorganized it is wiser to advise institutionalization as one old person may become a wasteful and debilitating burden to an entire family group It is often very difficult to bring the family to *hospitalize older people for understandable emotional reasons* though it is obvious that the patient would be better managed and happier in the objective atmosphere of a properly equipped hospital

The old person who is kept at home and whose thinking processes are clouded is actually made unhappy in the presence of those with whom he has had previous association at a higher level of intellectuality The contrast keeps him in a state of constant helpless frustration and adds immeasurably to suffering When such a one is placed in a quiet nursing home where he may enjoy comfortable solitude among contemporaries who have likewise passed their peak he lapses gratefully into a state of *torpor* which is infinitely more merciful and kindly than the feeling of competition in the home

POST-APOPLECTIC PSYCHOSES

Following cerebral accidents patients usually suffer mental deterioration and may become psychotic The management of these problems differs in no wise from that of the senile psychosis except that there may be such additional organic symptoms as pain urinary retention or incontinence

Course and Prognosis—The delirium usually lasts from three to six days Modern treatment has gone far to reduce the mortality which previously was as high as 10 per cent The greatest risks arise from complications especially infections and cardiorenal disease Amnesia for events occurring during the attack is usual

Treatment—The most urgent need is for fluid replacement and the administration of large amounts of vitamin B complex and salt A slow continuous intravenous drip of physiologic saline solution is set up and the soluble vitamins are introduced Sedation is used only when necessary and then judiciously As soon as possible a high caloric vitamin rich diet is given

Korsakoff Syndrome—Korsakoff syndrome is not limited to alcoholics but may be found in other toxic states and cerebral atherosclerosis In the alcoholic it commonly develops after an episode of delirium tremens

The characteristic change is the loss of memory for recent events retention of memory for remote events and a marked degree of confabulation The patient is disoriented especially as to time The mood is characteristically happy and expansive as reflected by the confabulation The patient confined to bed tells of luxurious parties plays he has seen in the company of elegant friends and so forth Insight is usually lacking There are usually associated peripheral neuritides ocular palsies and nystagmus

The acute symptoms usually last from four to six weeks Permanent residual damages are not however infrequent and may include memory defects and emotional and intellectual deterioration

Korsakoff's psychosis is treated in the manner of a delirium tremens Proper orthopedic care is given to prevent wrist and footdrop

Chronic Alcoholism—The chronic drinker who uses large amounts of alcohol develops a slow deterioration of the personality in all its spheres Intellectual keenness and perceptibility are dulled and the character is blunted especially in its ethical and esthetic aspects Psychosexual adjustments are especially liable to show deteriorating aspects such as promiscuity exposure and other sexual indecencies

The development of paranoid states is so common that some psychiatrists prefer a separate classification of *alcoholic paranoia* Psychologically this frequent association of alcoholism and paranoid delusions offers provocative spheres for speculation since the mechanisms involved in both conditions include the elements of repressed homosexuality and profound inferiority feelings Lying uncontrolled impulsive behavior personal carelessness and a deterioration of all social sense and responsibility are common The mood may vary between euphoria and depression with great irritability and hostility The usual lay portrait of the chronic alcoholic who is cruel and neglectful of his family but gay and charming when drinking with his fellow topers is fairly accurate

Treatment—Chronic alcoholism represents a chronic profound disturbance of the personality It requires *institutionalization* with the most thoroughgoing psychotherapy Ambulatory treatment is to no avail since short periods of abstinence are followed almost inevitably by recurrences

Psychoanalysis conducted in the protective confines of a suitable hospital is claimed to be helpful in perhaps 40 per cent of cases The results

found and lasting unconsciousness In the acute stages sometimes within twenty four to forty eight hours of the exposure to the gas a severe delirium may occur In other cases, a free period of several weeks may follow the comatose period before the onset of mental symptoms such as confusional states memory defects motor paralyses sensory changes and speech disturbances Rarely do intellectual and emotional defects persist

ALCOHOLIC PSYCHOSES

The general problem of alcoholism is elsewhere discussed (p 1385) There seems little doubt that the alcoholic begins his career as one suffering from a basic and profound personality disturbance Following the inordinate use of alcohol there result metabolic disturbances such as dehydration salt depletion and vitamin deficiencies which complicate the symptomatologic picture and contribute greatly to the clinical manifestations

Certain distinct clinical entities are recognizable in the alcoholic psychoses These include delirium tremens the Korsakoff syndrome chronic alcoholism acute alcoholic hallucinoses and pathologic intoxication Psychotic manifestations that persist after alcohol has been stopped may represent avitaminoses and should be treated appropriately with thiamine niacin and riboflavin (p 616)

Delirium Tremens —Delirium tremens is rarely encountered in patients under thirty It usually follows a prolonged unaccustomed debauch but may be seen in chronic alcoholics It may be precipitated by injury or intercurrent infection It was previously believed that delirium tremens could be induced in a chronic drinker by the sudden withdrawal of alcohol (so called abstinence delirium') but this is probably not true In recent years more emphasis has been laid on the etiologic role of metabolic physicochemical and vitamin factors though the etiology is still obscure

Clinical Manifestations —The delirium is usually preceded by sleeplessness and anorexia heightened irritability terror dreams and a growing feeling of apprehension The patient is increasingly fearful and starts at a trifling unexpected sound The delirium is usually intense visual hallucinations are characteristic such as seeing snakes and repulsive animals and tactile (vermin crawling on the skin) and occupational hallucinations are common The mood is generally one of terror although occasionally the patient is exhilarated expansive and in exaggerated good humor The patient is usually confused and disoriented in all spheres Misidentification of people is common and attention is most difficult to maintain Extreme suggestibility is usually present especially as to sensory impressions The patient is restless cannot sleep and is difficult to keep in bed

Physical Examination —Physical examination usually reveals marked inanition and dehydration The breath is foul and the lips are covered with crusts The face is flushed the lips dry and the pupils widely dilated Tremors of the hands accentuated on intention of the tongue lips and perioral muscles are present Speech is thick and indistinct The temperature is elevated and the pulse rapid and thready Albuminuria is common The nerve trunks and muscles may be tender Unless a complicating neuritis has abolished the deep tendon reflexes they are usually increased

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depend among other factors on the type of personality and the duration of the illness *Shock therapy* has proved valuable particularly when combined with psychotherapy The use of emesis as a *conditioned response* has been reported to 'cure about 40 per cent of a series of more than 800 patients observed for four years Large doses of vitamin B complex are given parenterally and orally for elimination of the deficiency factor

Acute Alcoholic Hallucinosis —The acute alcoholic hallucinosis is a state which usually follows excessive indulgence and consists of auditory hallucinations *accompanied by intense fear The voices usually are of an* accusing and threatening nature and their content reveals much of the unconscious conflict involved in alcoholism There may be olfactory and visual hallucinations as well

Illusions ideas of reference and misidentification are common Delusional states usually paranoid may develop The mood is one of fear apprehension and panic In these moods suicide is not infrequently attempted so that patients should be promptly *hospitalized*

Pathologic Intoxication —Pathologic intoxication occurs in a neurotic or psychopathic individual following the drinking of a trivial amount of alcohol The incident predicates more basic conflicts in personality It is distinguished from ordinary drunkenness by its greater severity and the pathologic nature of its manifestation The effect is one approximating an acute delirium The patient is confused hallucinated disoriented and has *temporary delusional ideas The mood is one of intense fear* and apprehension The upset is shortlived lasting rarely more than a day or two There is usually total amnesia following the episode

Treatment consists essentially of inducing sleep after which the attack is usually terminated Since this type of reaction is not a healthy way to attempt to deal with conflict the sobered patient is advised to undertake a more complete consideration of his personality difficulties under the guidance of the specialist

PSYCHOSES DUE TO SULFA DRUGS

Protracted and profound depressions with memory defects occasionally follow administration of sulfonamide drugs (p 94) It is wise to warn the patient concerning the possibility of the development of these reactions, which disappear more rapidly when *thiamine chloride* is injected *intravenously* in 100 *mg* doses or when *amphetamine sulfate* is administered orally

PSYCHOSES OCCURRING WITH MISCELLANEOUS DISORDERS

Psychoses are seen in a miscellaneous group of disorders such as multiple sclerosis hyperchromic anemia and epilepsy

Multiple Sclerosis —A number of patients with multiple sclerosis develop mental changes which are variable in type and severity (p 1504) Euphoria a lack of appreciation of the severity of the disease and a good natured acceptance of the affliction are fairly common Other patients are depressed and irritable with intellectual defects impairment of judgment and insight inattentiveness delusions and hallucinatory states

Hyperchromic Anemia —The mental symptoms of pernicious anemia may be apparent before the anemia is detectable From 5 to 35 per cent

of patients show minor mental disorders such as mood changes especially apathy and depression disinclination to work easy fatigability mild delirium paranoid states and intellectual deterioration See p 1077

Treatment is directed to the underlying disease

Epilepsy —The psychiatric problems encountered in epilepsy include *psychic equivalents of the epileptic seizure such as sudden uncontrolled* outbursts of excitement and destructive behavior including homicide *There may be psychotic states characterized by hallucinations especially* auditory and delusions of persecution or of a religious nature Periods of *depression and ecstasy not necessarily alternating and fugues are fairly* common. Mental deterioration is by no means as common as usually be*lieved and may represent a* bromide intoxication *The treatment of* epilepsy is detailed elsewhere (p 1517)

PREGNANCY AND POST PARTUM PSYCHOSES

See pp 2647 and 2720

CHAPTER 66

THE INVOLUNTARY NERVOUS SYSTEM INTRODUCTION PHYSIOLOGY INTEGRATIONS

Phylogeny
Anatomy
Physiology
Nomenclature
Involuntary and Voluntary Nervous Systems
The Involuntary Nervous System and the Emotions
The Involuntary Nervous System and the Hormones
The Involuntary Nervous System and Metabolism
The Involuntary Nervous System and Allergy
The Involuntary Nervous System and Visceral Controls
The Involuntary Nervous System and Psychosomatic Disease
Methods of Examination
Pharmacology of the Involuntary Nervous System
Surgery of the Involuntary Nervous System

Phylogeny —The cells of the involuntary nervous system arise from the cerebrospinal axis Phylogenetically the involuntary nervous system makes its first appearance in the anneld *the first phylum in which a vascular system is developed Of the* cells of the primitive involuntary nervous system one group retains as its function the transmission of nerve impulses the other develops into chromaffin tissue and acquires secretory activity In higher vertebrates chromaffin cells collect in the adrenal medulla while nerve cells retain their segmental arrangement

Anatomy —The elements of the involuntary nervous system are represented in the cortex diencephalon medulla cord thoracolumbar and craniosacral regions and in the peripheral ganglia and autonomic plexuses

Cortical Centers —The involuntary nervous system probably has its highest motor neurons in the *premotor cortex* More definitive centers are demonstrable in the *diencephalon (hypothalamus)* the *medulla* and the *spinal cord*

Hypothalamic Representation.—The *hypothalamic representation is suggested* in several clearly defined clinical disorders such a *paralysis agitans diabetes insipidus* and the *adiposogenital syndrome* The first of these as described by Parkinson is associated with many functional afflictions of the involuntary nervous system such as *salorrhea* and *oculogyric crises* With disproportionate frequency it coexists with *hyperthyroidism* and instances of *increasing exophthalmos* Diabetes insipidus and the adiposogenital syndrome are commonly regarded as disorders of the pituitary gland but experimental evidence points more clearly to a hypothalamic disorder in which the involuntary nervous system derangement initiates alterations in the metabolism of water fat deposition and gonadal activity

Medullary Centers —The medullary autonomic centers are clearly demonstrable Close to the midline beneath th sylvian aqueduct and the floor of the fourth ventricle there are nucl i which control *respiration salivation pupillary constriction vasomotor activity* and perhaps the metabolism of the *carbohydrat s*

Pathways of Cord —In the spinal cord the autonomic pathways traverse the *ventrolateral columns* This pathway is made up of ipsilateral and contralateral connections which cross in the brain *stem and at various spinal levels Some functions are* bilaterally represented while others seem entirely unilateral as in the instance of pupillary dilatation

Afferent Fibers —Visceral afferent fibers enter the cord in the posterior roots Their pathway follows the course of the other neurons which carry *pain* from other portions of the body Their cells lie in the posterior *horn* and the axons cross in the *anterior commissure*

to ascend in the opposite spinothalamic tract Practical application of this information is utilized in *anterolateral chordotomy* which abolishes most forms of visceral pain provided the section is carried down to the gray matter

The Thoracolumbar Division—The thoracolumbar division of the involuntary nervous system which is adrenergic is represented by a bilateral chain of twenty four *paravertebral ganglia* with their *rami communicantes* Each chain occupies a position lateral to the vertebral column and extends from the base of the skull to the *ganglion impar* at the coccyx

In the neck the ganglia connected with the three highest cervical nerves fuse to form the *superior cervical ganglion* The *middle cervical ganglion* is formed by a coalescence of the fourth and fifth cervical ganglia Just below this there is often an *intermediate ganglion* which is connected with the *inferior cervical ganglion* by two short fibers which encircle the vertebral artery

The inferior cervical and first thoracic ganglia are usually united into a single structure known as the *cervicothoracic or stellate ganglion* The remaining thoracic lumbar and sacral ganglia are much smaller and are subject to many anomalous variations Each however has a common arrangement relative to its communicating rami The *white ramus* which is *preganglionic* connects with the spinal nerves the *gray ramus* is *postganglionic* and passes out to the peripheral structures

The branches of the *upper five thoracic ganglia* are small and supply filaments to the thoracic aorta and its branches and to the bodies of the vertebrae and their ligaments Branches from the *lower seven ganglia* are large and unite to form the great lesser and small est splanchnic nerves The *great splanchnic nerve* is white and firm and is formed from the fifth sixth ninth and tenth ganglionic branches It terminates in the semilunar ganglion of the solar plexus and distributes filaments to the renal and suprarenal plexuses The *lesser splanchnic nerve* formed by filaments from the tenth and eleventh ganglia and the cord between pierces the diaphragm and joins the aorticorenal ganglion of the solar plexus It communicates in the thorax with the great splanchnic nerve and ends in the solar plexus The *smallest splanchnic nerve* arises from the last thoracic ganglion and pierces the diaphragm to terminate in the renal plexus

The Craniosacral Division—The craniosacral or cholinergic division is represented by the vagi and fibers in the *oculomotor facial* and *glossopharyngeal* cranial nerves Communications are also made with the *superior cervical ganglion* and the *carotid sinus plexus*

The vagus gives off the *recurrent laryngeal nerve* above the diaphragm within the thorax connections are established with the *inferior cervical ganglion* and the *cardiac pulmonary* and *esophageal plexuses* Below the diaphragm the *left vagus* sends branches along the lesser curvature to the anterior gastric wall and a large ramus to the *hepatic plexus* The *right vagus* sends fibers to the posterior gastric wall and through the celiac ganglion to the terminal plexuses in the upper abdominal viscera

The sacral representation leaves the spinal cord with the *second to fourth sacral nerves* in the cauda equina The *nervi erigentes* are formed and their postganglionic neurons connect with the intrinsic plexuses of the *genitalia bladder* and *rectum*

The Peripheral Autonomic Plexuses—The adrenergic and cholinergic fibers derived respectively from the thoracolumbar and craniosacral portions of the involuntary nervous system combine to form *peripheral plexuses* The most important of these are the *ciliary ganglia* associated with the oculomotor nerves the *superior cervical sympathetic ganglia* involved in the mechanism of pupillary dilatation and palpebral widening the *sphenopalatine ganglion* affected in Sluder s neuralgia the *carotid sinus nerve* which has highly specialized functions for the reflex control of blood pressure cardiac rate and respiration the *anterior and posterior cardiac plexuses involved in the mechanism of bronchial asthma the plexuses of Auerbach and Meissner* in the intestines the *phrenic adrenal renal splanchnic ovarian gastric hepatic splenic* and *superior mesenteric plexuses* which connect with the dorsal vagal ganglia *inferior mesenteric superior hypogastric* and *inferior hypogastric plexuses involved with the functions of the lower bowel the bladder and the genitalia*

The Ganglionic Relay Stations—In the involuntary nervous system the effector cell occupies a position outside the cord to which it is connected by a *preganglionic medullated fiber* The migrated cell gives origin to the true effector fiber which is *nonmedullated* and *postganglionic*

Whereas the impulse in the voluntary nervous system may pass from the cerebrospinal axis to the end organ without interruption in the involuntary nervous system *it is relayed at the peripheral ganglion* The accessibility of the relay station and its pre and post

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Cholinergic Division—The cholinergic subdivision is also known as *craniosacral* from its anatomical characteristics and *parasympathetic* in contrast to sympathetic

Adrenergic Division—The adrenergic subdivision is variously described as *thoracolumbar* or *sympathetic* The former label, which contrasts to craniosacral is dependent upon the anatomical distribution of its fibers the term sympathetic deserves to be eliminated since it is used by some writers to refer to the involuntary nervous system as a whole and by others in reference to the adrenergic subdivision

Involuntary and Voluntary Nervous Systems—In broad terms the voluntary nervous system is concerned with adaptation of the organism to its external environment the more primitive involuntary nervous system regulates the adaptation to internal environment Each of these adaptations is dependent upon influenced by and responsive to changes that occur in the other Phylogenically the involuntary nervous system is older and more primitive hence it is likely that the psyche is more influenced by the involuntary nervous system than vice versa

Physiologically the clinician observes frequent examples of interplay between *psyche* and involuntary nervous system Hunger contractions of the stomach register as appetite and initiate the desire to seek food but it is equally possible for the odor of fried bacon and freshly roasted coffee to set up hunger contractions in the stomach by way of the olfactory nerve and cerebral association tracts

Though the anatomical sites are not clearly defined there is incontrovertible evidence to attest to *supramedullary representation* of the involuntary nervous system In diseases characterized by *destruction of the cortex* there occur the symptoms of *adrenergic hypothalamic activity such as the increased muscle tonus rigidity flushing and tremor of paralysis agitans* (p 1505) *destruction of the hypothalamus* is associated with profound changes in the metabolism of water (*diabetes insipidus*) in the deposition of fat and gonadal activity (*adiposogenital syndrome*) and under experimental conditions at least *peptic ulcers* may be produced

The Involuntary Nervous System and the Emotions—The relationship between emotions and involuntary nervous system has been described in detail in the consideration of *neuroses* (p 1335) Overt or occult emotions are productive of an *autonomic imbalance* and conversely disturbances of the involuntary nervous system are accompanied by emotional overtones

Stimulation of the involuntary nervous system by the common emotions is everyday experience *Fear causes us to blanch or blush* we experience palpitation tachycardia or elevation of blood pressure we note *a desire to urinate or retention of urine* some observe movement of the bowels while others experience obstinate constipation there may be manifest rapid respirations or a spell of breath holding Whatever the *individual pattern* may be the syndrome is effected through the mediation of the involuntary nervous system

The opposite face of the coin reveals that autonomic imbalance is productive of emotional disturbances An injection of *epinephrine* sufficient to cause tachycardia and elevation of blood pressure gives rise to the sensations of anxiety and fear clinicopathologically the patient with *hyperthyroidism* (p 1197) is in constant emotional turmoil whether due

ganglionic fibers make possible the surgical approach to problems in which the involuntary nervous system is involved

Physiology—Physiologically the voluntary and involuntary nervous systems differ in that the former is influenced by the will and consciousness whereas the latter operates involuntarily and subconsciously The economy of this mechanism must be apparent to the practitioner when he realizes that the vital functions of regulation of heart beat, blood pressure digestive activity and the like proceed during sleep and without trespass upon the conscious functions of the cerebrum Dual innervation, in which both voluntary and involuntary elements participate is required for the socially modified visceral functions of urination defecation and sexual activity Repression of these instinctual propensities is important in the Freudian concept of the neuroses

The Subdivisions of the Involuntary Nervous System—The involuntary nervous system is best divisible according to its pharmacological reactions into adrenergic and cholinergic portions An explanation of the subdivisions has been suggested by Gaskell It is his hypothesis that the ancestor of the vertebrate presented a double segmentation appendiceal and branchial The *branchial segmentation* has given rise to the structures of the primitive endoderm and is concerned mostly with the metabolic functions of gastro-intestinal and respiratory tracts These structures receive augmentor impulses over the cholinergic system, which is stimulated by acetylcholine and inhibitory impulses through the adrenergic system whose stimulant, epinephrine depresses their functional activity

The *appendiceal segmentation* has given rise to the appendages of the skin the subdermal layer of musculature the vascular system and the derivatives of the segmental ducts from which arise the organs of excretion and reproduction Essentially these are concerned with self preservation they receive excitatory impulses via adrenergic system and are inhibited by cholinergic structures

The Tonicity of the Involuntary Nervous System—The involuntary nervous system is in tonic activity The subdivisions are mutually antagonistic and reciprocal in their innervation Paralysis of either one causes overplay as the result of the release of the antagonist.

It has been an easy assumption that *acetylcholine* regulated the normal tonic activity of the cholinergic system and that *epinephrine* functioned similarly for the adrenergic system However the physiological proof of this attractive theory has proved to be more difficult than its enunciation the presence of significant amounts of circulating acetylcholine remains to be proved and epinephrine is not present in the circulating blood in quantities that are anywhere near capable of tonic activity

Emergency Theory—It was the failure of proof of the tonicity theory that led to the substitution of the emergency theory of Cannon This postulated that in times of stress, epinephrine was poured forth to stimulate the adrenergic system This ingenious theory has not been substantiated since it appears that the amount of epinephrine that must be secreted, in order to produce an emergency effect, is beyond the secretory capacity of the gland. Those who have been most concerned with the physiology of the involuntary nervous system have been forced to admit that the theories of tonic or emergency control by known chemical agencies remain to be proved These discussions have more than academic importance as illustrated by surgical attempts to apply the epinephrine theories in practice For example it was the hope that denervation of the adrenal medulla or an almost complete bilateral adrenalectomy might control *hypertension* and *hyperthyroidism* These surgical efforts were unsuccessful as might have been predicated by physiologists more familiar with the fundamental facts

Nomenclature—The involuntary nervous system as a whole is sometimes labeled *autonomic vegetative* or *sympathetic* We prefer the use of the term involuntary since it emphasizes the important physiological differentiation between it and the neurologic mechanisms that are under the control of the will

The subdivisions of the involuntary nervous system are classified by us as *cholinergic* and *adrenergic* This pharmacophysiological terminology refers to the most characteristic features of each of the subdivisions the one is stimulated by *acetylcholine* and drugs of similar activity, the other responds to the effects of *epinephrine*

Cholinergic Division—The cholinergic subdivision is also known as *craniosacral* from its anatomical characteristics and *parasympathetic* in contrast to sympathetic

Adrenergic Division—The adrenergic subdivision is variously described as *thoracolumbar* or *sympathetic* The former label, which contrasts to craniosacral is dependent upon the anatomical distribution of its fibers the term sympathetic deserves to be eliminated since it is used by some writers to refer to the involuntary nervous system as a whole and by others in reference to the adrenergic subdivision

Involuntary and Voluntary Nervous Systems—In broad terms the voluntary nervous system is concerned with adaptation of the organism to its external environment the more primitive involuntary nervous system regulates the adaptation to internal environment Each of these adaptations is dependent upon influenced by and responsive to changes that occur in the other Phylogenically the involuntary nervous system is older and more primitive hence it is likely that the psyche is more influenced by the involuntary nervous system than vice versa

Physiologically the clinician observes frequent examples of interplay between *psyche* and involuntary nervous system Hunger contractions of the stomach register as appetite and initiate the desire to seek food but it is equally possible for the odor of fried bacon and freshly roasted coffee to set up hunger contractions in the stomach by way of the olfactory nerve and cerebral association tracts

Though the anatomical sites are not clearly defined there is incontrovertible evidence to attest to *supramedullary representation* of the involuntary nervous system In diseases characterized by *destruction of the cortex* there occur the symptoms of *adrenergic hypothalamic activity* such as the increased muscle tonus rigidity flushing and tremor of paralysis agitans (p 1505) *destruction of the hypothalamus* is associated with profound changes in the metabolism of water (*diabetes insipidus*) in the deposition of fat and gonadal activity (*adiposogenital syndrome*) and under experimental conditions at least *peptic ulcers* may be produced

The Involuntary Nervous System and the Emotions—The relationship between emotions and involuntary nervous system has been described in detail in the consideration of *neuroses* (p 133a) Overt or occult emotions are productive of an *autonomic imbalance* and conversely disturbances of the involuntary nervous system are accompanied by emotional overtones

Stimulation of the involuntary nervous system by the common emotions is everyday experience Fear causes us to blanch or blush we experience palpitation tachycardia or elevation of blood pressure we note a desire to urinate or retention of urine some observe movement of the bowels while others experience obstinate constipation there may be manifest rapid respirations or a spell of breath holding Whatever the *individual pattern* may be the syndrome is effected through the mediation of the involuntary nervous system

The opposite face of the coin reveals that autonomic imbalance is productive of emotional disturbances An injection of *epinephrine* sufficient to cause tachycardia and elevation of blood pressure gives rise to the sensations of anxiety and fear clinicopathologically the patient with *hyperthyroidism* (p 1197) is in constant emotional turmoil whether due

to direct action resulting from the metabolic disturbance or through a conditioned reflex or emotion

The 'compleat practitioner" must reckon with the *visceral and somatic representations of emotion* and the *emotional overtones of visceral and somatic activities* In each instance the mediation is by way of the involuntary nervous system

The Involuntary Nervous System and the Hormones—Hormonal activity is admittedly under the principal control of chemical and metabolic influences Many clinical observations point to the additional significance of neurogenic mechanisms in the total economy the manifestations of *hyperthyroidism* are obviously sympathomimetic *diabetes insipidus* is frequently initiated by psychogenic factors, fear is often accompanied by *glycosuria* and precedes the onset of many instances of *diabetes mellitus* (p 1246), whose intensity it may markedly increase the syndrome of *anorexia nervosa* is almost indistinguishable from that of total destruction of the anterior pituitary gland (*Simmonds disease*) (p 1169)

The Involuntary Nervous System and Metabolism—There are reciprocally important interrelationships between involuntary nervous system and metabolic phenomena, injections of *epinephrine* produce glycosuria and hyperglycemia the administration of *iodide* results in a striking amelioration of the symptoms of hyperthyroidism (p 1197) *myxedema* is associated with profound changes in subcutaneous tissues lesions involving the *hypothalamus* are characterized by abnormalities in water balance and deposition of fat the rate of *oxygen consumption* and the tempo of *energy metabolism* are strikingly augmented by *sympathomimetic activities* which also increase muscle tone and metabolism

The Involuntary Nervous System and Allergy—The importance of the involuntary nervous system in the pathogenesis of allergy is best illustrated by the relief afforded to *atopic phenomena* (p 547) by injections of *sympathomimetic amines* such as epinephrine angioneurotic phenomena abate the bronchial musculature of the asthmatic relaxes the turgid nasal mucous membrane shrinks and the congested conjunctiva blanches

The Involuntary Nervous System and Visceral Controls—Each tissue or viscus is under the control of the involuntary nervous system to greater or lesser degree *Coordination* is assured through dual and reciprocal innervations transmitted through adrenergic or cholinergic subdivisions which function in a manner that is mutually antagonistic Positive reactions represent the sum of stimulation of stimulator and inhibition of inhibitor negative responses follow inhibition of stimulator and stimulation of the inhibitor In any given instance the end plate may be motor or secretory but the general principle of innervation is uniform

These broad tenets are illustrated more concretely in the digestive and circulatory systems At the sight of food digestive secretions and peristaltic gradient are initiated The bolus is carried down through the esophagus by alternate waves of contraction and relaxation at the sphincters relaxation permits the passage of food through the tract where it is attacked by digestive juices and churned by muscle activity the release of secretin initiates the flow of bile intestinal and pancreatic juices so that these are in readiness for the aliment after it has passed pyloric sphincter when thorough absorption of nutritive material has been ac-

complished and the fecal column reaches the rectum the voluntary nervous system is notified lastly at a proper time and place the stool is expelled as the result of the combined efforts of voluntary contractions of abdominal muscles and involuntary relaxation of the rectal sphincter Consistent with the symphonic concept of involuntary nervous system activities these alimentary phenomena are not solo parts the tissues in which active digestion is occurring require and receive additional quantities of blood through local vasodilatation and engorgement the cardiac rate speeds appreciably during the peak of digestive activity splanchnic engorgement is accomplished at the expense of the efficiency of the cerebrum and voluntary muscles as evidenced by the somnolence and lethargy that follow a hearty meal

Cardiovascular coordinations are equally remarkable Neurogenic or psychogenic stimuli may produce narrowing of the caliber of the arteries blood pressure then rises as the result of which the cardio inhibitor centers effect a compensatory bradycardia To the contrary with dilatation of the peripheral blood bed the systemic blood pressure falls while a central action initiates a compensatory tachycardia Each physiological change is capable of setting in motion registers and brakes of the greatest delicacy so that the total human economy operates silently flawlessly and with incredible efficiency

The Involuntary Nervous System and Psychosomatic Disease—The mutual relationships that exist between psyche voluntary and involuntary nervous system have been previously emphasized Physiologically the voluntary and involuntary nervous systems differ in that the former is influenced by the will and consciousness whereas the latter operates involuntarily and subconsciously For the most part the involuntary nervous system is the ubiquitous and silent partner The more the voluntary nervous system is freed from the insubordinate involuntary system consistently with the harmonious interaction between the systems the greater the advantage to the animal (Gaskell) There are however limits to the tolerance and durability of the involuntary nervous system the demands of civilization may progress (or regress) to the point that this primitive tissue can no longer remain silent under existing conditions It protests by the minor and usually inconsequential symptoms of the syndrome of *autonomic imbalance* (p 1395) More severe or protracted irritation leads to *somatization* and the production of *demonstrable tissue lesions* elsewhere described (p 1344)

Methods of Examination—The involuntary nervous system does not lend itself readily to methods of laboratory investigation The surgeons who have been confronted with the problem of estimating the influences of the involuntary nervous system have devised ingenious indirect methods for approximating the activities of the system relative to the peripheral vasculature and the vasodermal structures

Measurements of *skin temperature blood flow* and *limb volume* are noted before and after *procaine block general anesthesia* and the *intravenous injection of foreign protein* (typhoid vaccine) *oscillometric studies* are made of the accessible arteries of the extremities By indirection an assumption is valid regarding the tonus of the involuntary nervous system as a whole this serves as an index for the probable success of surgical

procedures particularly in hypertension (p 900) and vasospastic disorders (p 791)

Pharmacology of the Involuntary Nervous System—The specific pharmacologic agents whose activities are concerned primarily with the involuntary nervous system include

Cholinergins Acetylcholine, mecholyl doryl pilocarpine physostigmine neostigmine See p 3873

Depressants of the Cholinergic System Belladonna atropine scopolamine homatropine eucatropine novatropine syntropan trasentin See p 3875

Adrenergens Epinephrine ephedrine amphetamine (Benzedrine) neo synephrine, propadrine, paredrine privine kephrine See p 3876

Inhibitors of the Adrenergic System Ergotamine ergotoxine nicotine See p 3883

Table 90—Surgery of the Involuntary Nervous System

Procedure	Indication
Resection of superior cervical sympathetic ganglion	Facial paralysis for closure of eye (p 1484) Angina pectoris for relief of pain (p 890)
Denervation of carotid sinus	Carotid syncope (p 992)
Stellate ganglionectomy	Angina pectoris Neuralgias and causalgias of arm or amputation stump Raynaud s disease of arm (p 1000) Hyperidrosis of arm
Preganglionic upper thoracic sympathectomy	Vasospastic disorders of arms
Vagectomy	Peptic ulcer (p 1780)
Supra and infra-diaphragmatic splanchnicectomy	Essential hypertension (p 900) Congenital megacolon (p 1871)
Paravertebral alcohol injection of thoracic ganglia or lumbar ganglia	*Cardio aortic pain abdominal pain*
Lumbar sympathectomy	Vasospastic disorders of legs spastic paralysis
Presacral neurectomy	Relief of pain
Peri arterial sympathectomy	Raynaud s disease (p 1000)
Peripheral sympathectomy	Peripheral endarteritis (p 994) Thrombo angiitis obliterans (p 1009)

Surgery of the Involuntary Nervous System—The surgical approach to the involuntary system constitutes one of the outstanding achievements of modern times It is quite likely that the accomplishments are as yet only in their first phases but the summary in Table 90 indicates present potentialities

CHAPTER 67

THE INVOLUNTARY NERVOUS SYSTEM PARTICIPATION IN CLINICAL DISTURBANCES

The wide scope of the activities of the involuntary nervous system predicates its participation in the majority of clinical disturbances that affect the human mechanism. The most important of these are indicated in Table 91.

PRIMARY DISTURBANCES OF THE INVOLUNTARY NERVOUS SYSTEM

In addition to the participation of the involuntary nervous system in many systemic disturbances, seemingly primary phenomena are encountered. Most often, patients suffer the widespread functional disorder of *autonomic imbalance* (p. 1395); less often, the affliction is more localized, as in *carotid sinus syncope* (p. 922), the *vasospastic diseases* and the *trophoderms*.

Vagotonia and Sympathicotonia

We do not subscribe to the existence of the distinct clinical entities of vagotonia and sympathicotonia. In the former, the patient is believed to exhibit the symptoms that are equivalent to an injection of an effectual dose of a cholinergic drug such as physostigmine, with resultant bradycardia and intestinal spasmosis. In sympathicotonia, there are the manifestations that mimic the results of an effectual injection of epinephrine, with tachycardia, elevation of blood pressure, nervousness and palpitation. Unfortunately for purposes of classification, pure vagotonia and pure sympathicotonia are never demonstrable; most patients exhibit evidences of aberrations in the tonus of the involuntary nervous system which we have labeled "autonomic imbalance."

Autonomic Imbalance

In his everyday experience the practitioner encounters widespread disturbances in the realm of the involuntary nervous system. Some of the phenomena are completely subjective (nervousness, asthenia), while others, though lacking evidences of organic disease, are objectively apparent.

TABLE 91.—PARTICIPATION OF INVOLUNTARY NERVOUS SYSTEM IN CLINICAL DISTURBANCES

Infection	
Typhoid fever	Febrile bradycardia and lymphocytosis
Tuberculosis	Adrenergic flush sweat and tachycardia beyond febrile effect
Allergy	
Urticaria	Relieved by epinephrine
Angioneurotic edema	Relieved by epinephrine
Vasomotor rhinitis	Relieved by epinephrine
Bronchospasm	Relieved by epinephrine
Metabolism	
Hyperglycemia	Produced by epinephrine
Elevation of basal metabolic rate	Produced by epinephrine
Increased oxygen consumption	Produced by epinephrine
Glycosuria	Produced by epinephrine and by injury to central nervous system
Water exchange	Polyuria and polydipsia from lesions of hypothalamus (diabetes insipidus)
Fat deposition	Obesity and genital dystrophy from lesions of hypothalamus (Frohlich s syndrome)
Muscle metabolism	Tremors produced by epinephrine with increased excretion of creatinine
Circulation	
Blushing	Atropine effect
Blanching	Epinephrine effect
Vasospasm with cold extremities to point of gangrene	Ergot poisoning sensitivity to overdosage with ergotamine nicotinism (?) Raynaud s disease (p 1000) with operative relief from sympathectomy
Angiospasms	Produced by epinephrine and nicotine relieved by procaine block and sympathectomy (p 91)
Angina pectoris	Produced by epinephrine relieved by sympathectomy (p 890)
Hypertension	Produced by epinephrine relieved by sympathectomy (p 900)
Bradycardia	Produced by cholinergins (physostigmine) relieved by atropine
Tachycardia	Produced by epinephrine nicotinism (?)
Coronary dilatation	Follows sympathectomy (?)
Cardiac irregularities	Produced by epinephrine relieved by sympathectomy (p 873)
Raynaud s disease	Simulated by ergotism relieved by sympathectomy
Erythromelalgia	Peripheral vascular neurosis"
Acroparesthesia	Peripheral vascular neurosis"
Trench Foot	Peripheral vascular disturbance
Thrombo-angiitis obliterans	Partially simulated by ergotism accentuated by smoking (nicotine)
Blood	
Lymphocytosis	Produced by epinephrine
Eosinophilia	In allergies

TABLE 91—PARTICIPATION OF INVOLUNTARY NERVOUS SYSTEM IN CLINICAL DISTURBANCES (*Continued*)

Endocrine System	
Hyperthyroidism	Preceded by autonomic imbalance simulated by epinephrine lymphocytosis
Diabetes insipidus	Posterior pituitary or hypothalamic lesion
Adiposogenital syndrome (Frohlich)	Pituitary or hypothalamic lesion
Adrenal cortical deficiency (Addison)	Relieved symptomatically by epinephrine
Status thymico-lymphaticus	Lymphocytosis
Diabetes mellitus	Simulated by injections of epinephrine inhibited by pituitary removal and injury to hypothalamus (Houssay)
Neurology	
Autonomic imbalance	Functional disturbances usually on constitutional or emotional basis often with neurosis (p 133)
Psychosomatic disease	Autonomic imbalance (p 139) with end-organ pathology (p 1344) conversion hysteria (p 13 3)
Epilepsy	Explosive autonomic seizures (?)
Migraine	Dilatation of cerebral vessels
Headache	As migraine (p *1509*)
Paralysis agitans	Central autonomic lesion occasionally with exophthalmos and hyperthyroidism
Hypothalamic disease	See Pituitary Disorders (p 11 ?)
Respiration	
Vasomotor rhinitis	Allergy relieved by epinephrine
Bronchial asthma	Allergy relieved by epinephrine
Digestion	
Secretory disturbances	See Gastric Secretory Neuroses (*p 1 67*)
Motor disturbances	See Cardiospasm (p 1 24) Gastric Motor Neuroses (p 1 6) Peptic Ulcer (p 1 30) Pylorospasm Regional Ileitis (p 1851) Mucous Colitis (p 1846) Intestinal Neuroses (p 1845) Nonspecific Ulcerative Colitis (p 18 6) Sigmoid Spasm (p 1846) Congenital Megacolon (p 1871)
Urinary System	
Polyuria	See Hypothalamus (*p 13 5*)
Bladder innervation	See Retention of Urine (p 2 61) Incontinence of Urine (p 2 65)
Reproductive Systems	
Uterus	See Dysmenorrhea (p 2561) Parturition (p 26 7)
Genitalia	See *Erection (p 240)* Orgasm (p 2490) Frigidity (p 2491) Impotence (p 2499)
Climacteric	Male (p 2412) and female (p 2 3) associated with manifestations of autonomic imbalance

TABLE 91 —PARTICIPATION OF INVOLUNTARY NERVOUS SYSTEM IN CLINICAL DISTURBANCES (*Continued*)

Skeletal and Locomotor	
Tremors	Produced by epinephrine partially inhibited by atropine in hyperthyroidism and paralysis agitans
Muscle dystrophies	Myasthenia gravis (p 2886) relieved by physostigmine (p 2886)
Tegumentary	
Hyperidrosis	Produced by epinephrine
Pilomotor activity	Produced by epinephrine
Cutaneous allergies	Relieved by epinephrine
Cutaneous vascular disorders	Simulated by epinephrine
Cutaneous thermal disorders	Simulated by epinephrine
Eye	
Mydriasis	Produced by instillations of atropine and epinephrine
Miosis	Produced by instillations of pilocarpine and eserine
Enophthalmos	Produced by paralysis of superior cervical sympathetic ganglion
Exophthalmos	Produced by hyperthyroidism through unknown mechanism

(flushing blushing tachycardia diarrhea lability of the blood pressure motor and secretory disturbances of the intestinal tract sweating or tremor)

Autonomic imbalance is a vexing and irritating condition so far as clinical practice is concerned Obviously its production is closely allied to emotional instability the practitioner recognizes that many if not all of its manifestations are seen in the *anxiety neuroses* (p 1347) An understanding of the condition has not been furthered by the dismissal of these complaints as functional neurotic or hypochondriacal or as evidences of larval hyperthyroidism formes frustes of hyperthyroidism basedoid or manifestations of adrenal insufficiency

Etiology —The factors that *predispose* to autonomic imbalance are unknown Most patients note some manifestation as far back as memory exists Certainly the predisposition seems to be inherited or associated with some early environmental factor

The *exciting causes* of autonomic imbalance are more clearly delineated They occur most often during the era of changes in sexuality and are more common at puberty at the menopause and during menstruation More directly however autonomic imbalance is precipitated by *psychic insults* and is associated with the various neuroses (p 1335)

Clinical Manifestation —The clinical manifestations of autonomic imbalance are varied They include palpitation shortness of breath cardiac irregularity particularly premature contractions headache insomnia attacks of diarrhea intestinal spasmoses secretory disturbances of the stomach and intestines menstrual disturbances particularly dysmenorrhea excessive sweating vasomotor instabilities such as blushing flush

ing or coldness of the extremities emotional instability tachycardia bradycardia tremor nervousness and asthenia

The Individual Reaction Picture—Despite the variety in the manifestations of the functional disturbances of the involuntary nervous system each individual has a unique reaction picture Those who suffer from pylorospasm always have pylorospasm and those with tachycardia never develop bradycardia The reaction picture never really disappears but is subject to exacerbations and remissions At any given time it can be activated by fatigue and emotional disturbances such as fear fright anxiety or anger

Laboratory Data—The physical examination of the patient with autonomic imbalance is disturbingly normal With intestinal symptoms roentgenograms reveal only functional disturbances such as excessive motility or spasm gastric test meals show only secretory and motor changes The basal metabolic rate is normal though the clinical symptomatology suggests a hyperthyroidism blood counts occasionally reveal a tendency towards lymphocytosis and an unusual sensitivity is observed to the subcutaneous injections of epinephrine The last previously called the Goetsch test was regarded as pathognomonic of excessive activity of the thyroid gland and an indication for surgical procedures Its nonspecificity is apparent from the fact that at least 33 per cent of normal medical students show the identical response

Diagnosis—The diagnosis of autonomic imbalance is established by exclusion Since adrenergic symptoms simulate hyperthyroidism the estimation of the basal metabolic rate and an adjudication of the response to iodide are warranted before a final opinion is expressed Those patients with gastro intestinal symptoms are entitled to stool examinations and contrast radiography since the manifestations strongly suggest peptic ulcer in many instances

Course—The vast majority of patients with autonomic imbalance learn to live with their functional affliction without recourse to medical therapy Others in association with the *conversion neuroses* (p 1363) progress to the stages of organic disease The *psychosomatic manifestations* which in our opinion are related to the conversion mechanism in hysteria (p 1353) are elsewhere described in detail (p 1344) It is sufficient to state here that they have the three components of psychogenic autonomic and end organ manifestations The last of these is unquestionably a result of the operation of the first two elements Which of these latter precedes the other remains a problem as difficult of solution as that of the determination of the chicken egg sequence

Treatment—The first essential factor in the management of autonomic *imbalance is assurance* that the patient has no organic disease The practitioner must then find time to enter into an explanation of the nature of the disturbance its chronicity its pathologic importance and its relationship to emotional problems

See *Nontechnical Psychotherapy* (p 1310)

Attempts at *specific therapy* usually result in failure Such formidable operations as subtotal thyroidectomy denervation of the adrenal medulla and partial adrenalectomy are ill advised and carry inordinate risk

Specific *pharmacotherapy* is also disappointing except in the instance

TABLE 91—PARTICIPATION OF INVOLUNTARY NERVOUS SYSTEM IN CLINICAL DISTURBANCES (Continued)

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ness or engorgement of the veins An attack may last for one to several days There is no present available form of effectual therapy

NEOPLASMS

On rare occasions *chromaffin tumors* are seen in the structures of the involuntary nervous system These include *neuroblastomas* *paraganglioma*s and *ganglioneuromas* They are discussed in greater detail with the disturbances of the *adrenal medulla* (p 1263)

14

of the control of excessive vagal activity by the belladonna derivatives Sedation with barbiturates and the use of hypnotics at bedtime tend to take off a good deal of strain Attempts to regulate the involuntary nervous system by administering hormones particularly those derived from the sex glands are capable of much more harm than good

Diencephalic Autonomic Seizure

A rare reaction pattern of the hypothalamus is the diencephalic autonomic seizure which consists of *convulsive attacks* of *aura* and *loss of consciousness* The aura is usually a *bitemporal headache* This is followed by loss of consciousness during which there is a sudden diffuse *flushing* or *pallor* of the skin the appearance of a *macular rash* or an *urticaria* on one or both sides of the body The pupils are widely dilated and fluctuate in size, excessive tearing sweating and salivation are noted

Penfield believes that the reaction pattern is hypothalamic and occurs in association with tumors or affections of the floor of the third ventricle or neoplasms in the region of the sella The syndrome is specialist province and the patient is referred to the neurosurgeon for more complete study and exploratory craniotomy if necessary

Carotid Sinus Syncope (Vasovagal Syncope)

At the region of the bifurcation the wall of the carotid artery contains a rich plexus of specialized sensory end plates By means of these the carotid sinus nerve joins the glossopharyngeus and the central nervous system With hyperactivity of the *carotid sinus* the patient notes unexplained attacks of *dizziness fainting* and *convulsions* During the seizure the blood pressure falls and the heart rate slows

Attacks of carotid sinus syncope are often precipitated by emotional upset the pressure of a tight collar digital pressure applied for purposes of stopping a cardiac irregularity the pressure of a tumor in the neck or an aneurysm of the vessel Often there is no apparent precipitating factor

Treatment is accomplished surgically by stripping the nerve plexus from the artery at the bifurcation or by section of the carotid sinus nerve The results of therapy are excellent See also p 923

Paralysis of the Cervical Sympathetic (Horner s Syndrome)

Paralysis of the cervical sympathetic may follow lesions of the medulla and pons but more often it is due to a tumor or a syringomyelia that involves the first thoracic and eighth cervical roots The Horner syndrome (spinal miosis) consists of *constriction of the pupil narrowing of the palpebral fissure enophthalmos pseudoptosis absence of the ciliospinal reflex flushing of the face and neck* and a local elevation of temperature The oculomotor nerve *is intact the pupils react to light and ptosis* can be voluntarily overcome

The syndrome is not amenable to therapy

Hereditary Edema of the Legs (Milroy s Disease)

Milroy s disease consists of a *chronic circumscribed edema* of both legs It appears soon after birth when a brawny swelling is noted without red

Electroencephalography —Perhaps the greatest advance in neurological diagnosis has been the introduction of electroencephalography This method records the electric potentials that originate from the brain and is similar to electrocardiography The electrodes are placed on the scalp by means

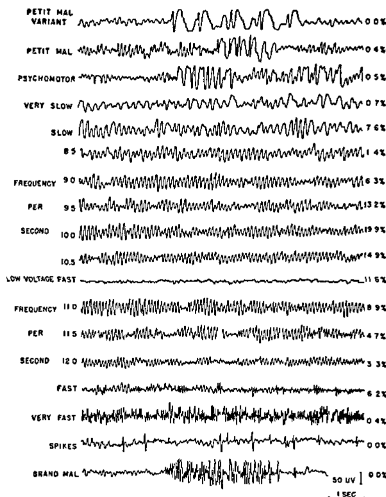


Fig 8 —Types of pattern encountered in electroencephalograms in adults The order of arrangement is slow at the top fast at the bottom The top three and bottom two patterns are classed as paroxysmal i e manifesting seizure discharges The names applied to each pattern are shown at the left The percentage incidence of each pattern in the normal control group appears at the right Time and voltage calibrations are given in the lower right corner *

of collodion It is rarely necessary even to cut the hair Through the electrodes the minute scalp currents are amplified by vacuum tubes until they activate an electromagnetic field whose oscillations are noted and written by a pen attached to a sensitive lever

* Gibbs in Manual of Military Neuropsychiatry by Solomon and Yakovlev

CHAPTER 68

THE VOLUNTARY NERVOUS SYSTEM METHODS OF DIAGNOSIS AND TREATMENT

CLASSIFICATION OF THE NEUROLOGIC DISORDERS

Because of the protection of nerve tissue by the bony covering of the cranium and vertebral column primary disorders are of infrequent occurrence the majority of *neurologic afflictions represent secondary responses* to vascular bacteriologic and metabolic derangements Many syndromes remain obscure and difficult of study and there is a disappointingly long list of purely descriptive conditions of unknown etiology and pathogenesis

The clinical disturbances are classified under the following main headings

- Congenital abnormalities
- Neoplasms
- Circulatory disorders
- Trauma and mechanical affections
- Infection
- Descriptive neurology to include nonbacterial inflammations the scleroses migraine and the epilepsies

NEUROLOGIC DIAGNOSIS

During his routine examination the practitioner notes mental attitudes and aptitudes speech oculomotor activities pupillary reflexes knee-jerks ankle-jerks abdominal reflexes nuchal rigidity motor strength visual acuity and the appearances of ear drums and fundi oculi On indication the deep reflexes are more carefully plotted and a sensory survey is inaugurated with reference to reactions to touch and pain (p 1476) The response to vibration is elicited over the tibias In the presence of positive findings or a suspicion of pathologic invasion particularly by micro organisms the practitioner performs lumbar puncture and has the spinal fluid examined for its cytology bacteriology serology and for the colloidal gold reaction (p 3734) Radiographs of skull and vertebral column furnish additional information

With positive findings neuropsychiatric consultation is mandatory for more refined studies which may include psychometry and electroencephalography The neurosurgeon is brought into the survey for ventriculography encephalography laminography and exploratory craniotomy or laminectomy The ophthalmologist assists by plotting the visual fields and ascertaining with greater certainty the changes in the fundus The otologist cooperates by charting the acuity of hearing and by performance of the vestibular reactions (p 2018)

Electroencephalography—Perhaps the greatest advance in neurological diagnosis has been the introduction of electroencephalography. This method records the electric potentials that originate from the brain and is similar to electrocardiography. The electrodes are placed on the scalp by means

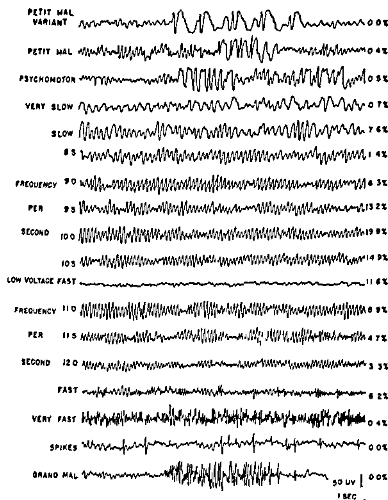


Fig 28.—Types of pattern encountered in electroencephalograms of adults. The order of arrangement is slow at the top, fast at the bottom. The top three and bottom two patterns are classed as paroxysmal, i.e., manifesting seizure discharges. The names applied to each pattern are shown at the left. The percentage incidence of each pattern in the normal control group appears at the right. Time and voltage calibrations are given in the lower right corner.

of collodion. It is rarely necessary even to cut the hair. Through the electrodes the minute scalp currents are amplified by vacuum tubes until they activate *an electromagnetic field* whose oscillations are noted and written by a pen attached to a sensitive lever.

[illegible] in Manual of Military Neuropsychiatry by Solomon and Yakovlev

The record an *electroencephalogram* is obtained under careful conditions with the patient lying down eyes closed A variety of brain waves" of various amplitudes and frequencies and shapes are observed Among the waves experts identify *alpha beta* and *delta* varieties which vary in frequency and voltage The records of various parts of the skull show differences in voltage and distribution but symmetrical points should give electroencephalograms that are almost identical

The normal electroencephalogram varies with emotional states the solution of mental problems movements of the eyelids sleep and the use of drugs Its greatest value is the altered pattern under pathological conditions such as *idiopathic epilepsy vascular pathology lacerations tumors* of the cerebral tissue and severe *dysfunctions* Localization of tumors of the convexity is made possible in this way

Classification of Electroencephalograms—Many variations are recognized in electroencephalographic tracings The following table based on Fig 258 (p 1403) briefly describes the more important variations with the exception of focal abnormalities which are depicted in Fig 273 (p 1427)

Tracing	*Comment*
Petit mal variant	Seizure discharges present Two per second with alternating wave and spike
Petit mal	Seizure discharges present. Three per second with alternating wave and spike
Psychomotor	Seizure discharges of flat or round topped appearance Record previously free from such activity
Very slow	Less than eight per second Twenty times more common in epilepsy Frequent in organic brain disease especially with convulsions (p 1519)
Slightly slow	Less than eight and one half per second Twice as common in epilepsy
Normal	From eight and one-half to twelve per second. No seizure discharges Normal trace appears in 15 per cent of epileptics
Low voltage fast	Less than 20 microvolts
Slightly fast	Greater than twelve per second Twice as common in epileptics
Very fast	Greater than twelve per second with large amount of activity Twenty times more common in epilepsy and frequent in organic brain disease especially with convulsions
Grand mal	Seizure discharges present Thirty three times more common in epileptics
Focal abnormalities	Abnormal activity in a particular cortical area, indicative of organic brain disease with localization (p 1425)

Reaction Patterns —Involvement of any particular part of the nervous system produces to a greater or lesser degree a specific reaction pattern which is the same whether the area has been traumatized infected infiltrated by neoplastic tissue degenerated or compressed Irritation of *sensory roots* produces neuralgias causalgias and paresthesias while destruction results in loss of sensation On the *motor side* irritations are manifested by fibrillations tremors contractions tics spasms choreiform movements athetosis and convulsions whereas complete destruction is followed by atrophy pareses and paralyses

Localization —The localization of the site of a neurologic lesion is deduced by correlating the abnormalities noted in sensation muscle movement and the reflex arc Lesions that involve a peripheral or [illegible]nal *sensory*

nerve are followed by anesthesia without paralysis wherea the contrary situation exists when a peripheral or cranial *motor nerve* is involved

Destruction of a *mixed nerve* is associated with combined motor and sensory phenomena By plotting the cranial or peripheral motor and sensory defects it should be possible to determine whether the pathway has been injured peripherally at the root segmentally in the cord along the pathways or in the supramedullary portions of the nervous system

Neurologic Consultation —Neurologic consultations are provided by the consultant organic neurologist and the neurosurgeon References to the latter are more satisfactory since the decreased morbidity and mortality of operative procedures have greatly widened the cope of surgery and *exploratory craniotomy* and *laminectomy* are often required in the clarification of puzzling neurologic syndromes

NEUROLOGIC THERAPY

Neurologic therapy is far from the confused and hopeless exercise of former times The roster presented below suggests the possibilities of both nonoperative and operative therapeutic measures

Pharmacotherapy —Useful drugs in the treatment of nervous and mental disorders include analgesic antipyretics sedatives and hypnotics anticonvulsants alcohol opiates demerol trichlorethylene cerebral stimulants general anesthetics arsenicals the sulfonamides and penicillin

Other Forms of Therapy —Besides drugs the practitioner may utilize for his neurologic patient the principles of rest restoration of function passive motion active motion exercise corrective exercise occupational therapy the vacation balneotherapy and spa therapy massage lumbar puncture cisternal puncture ventricular puncture intraspinal injection spinal anesthesia caudal anesthesia paravertebral nerve block physiotherapy by heat or cold baths electrotherapy and the electroconvulsive modalities

Specific Nonoperative Therapy

1 Sulfonamides and penicillin in the treatment of meningitis (p 443) and syphilis (p 1377)
2 Ketogenic diets and dehydration in epilepsy (p 1515)
3 Dilantin tridione and phenobarbital in epilepsy ergotamine in migraine (p 1507)
4 Antiluetic therapy in the syphilitic disorders (p 1377)
5 Vitamin administration in deficiency states (p 616)
6 Relief of trigeminal neuralgia by inhalations of trichlorethylene (p 148?)
7 Analgesia with salicylates (p 3833) the coal tars (p 3832) demerol (p 3863) the opiates (p 3853) and cobra venom (p 1049)
8 Treatment of flaccid paralysis by physiotherapy (p 3784)
9 Treatment of spastic paralysis by curare and similar preparations (p 3888)
10 Treatment of respiratory paralysis by the administration of oxygen containing 5 to 7 per cent of carbon dioxide or by a respirator (Iron Lung)
11 Treatment of medullary paralyses by picrotoxin as in poisoning by the barbiturates (p 3839)

The record, an *electroencephalogram* is obtained under careful conditions with the patient lying down eyes closed A variety of "brain waves" of various amplitudes and frequencies and shapes are observed Among the waves experts identify *alpha beta* and *delta* varieties which vary in frequency and voltage The records of various parts of the skull show differences in voltage and distribution but symmetrical points should give electroencephalograms that are almost identical

The normal electroencephalogram varies with emotional states the solution of *mental* problems movements of the eyelids sleep and the use of drugs Its greatest value is the altered pattern under pathological conditions such as *idiopathic epilepsy vascular pathology lacerations tumors* of the cerebral tissue and severe *dysfunctions* Localization of tumors of the convexity is made possible in this way

Classification of Electroencephalograms—Many variations are recognized in electroencephalographic tracings The following table based on Fig 258 (p 1403) briefly describes the more important variations with the exception of focal abnormalities which are depicted in Fig 273 (p 1497)

Tracing	*Comment*
Petit mal variant	Seizure discharges present Two per second with alternating wave and spike
Petit mal	Seizure discharges present Three per second with alternating wave and spike
Psychomotor	Seizure discharges of flat or round topped appearance Record previously free from such activity
Very slow	Less than eight per second Twenty times more common in epilepsy Frequent in organic brain disease especially with convulsions (p 1519)
Slightly slow	Less than eight and one half per second Twice as common in epilepsy
Normal	From eight and one-half to twelve per second No seizure discharges Normal trace appears in 18 per cent of epileptics
Low voltage fast	Less than 20 microvolts
Slightly fast	Greater than twelve per second Twice as common in epileptics
Very fast	Greater than twelve per second with large amount of activity Twenty times more common in epilepsy and frequent in organic brain disease especially with convulsions
Grand mal	Seizure discharges present Thirty three times more common in epileptics
Focal abnormalities	Abnormal activity in a particular cortical area indicative of organic brain disease with localization (p 1493)

Reaction Patterns —Involvement of any particular part of the nervous system produces to a greater or lesser degree a specific "reaction pattern" which is the same whether the area has been traumatized infected infiltrated by neoplastic tissue degenerated or compressed Irritation of *sensory roots* produces neuralgias causalgias and paresthesias while destruction results in loss of sensation On the *motor side* irritations are manifested by fibrillations tremors contractions tics spasms choreiform movements athetosis and convulsions whereas complete destruction is followed by atrophy pareses and paralyses

Localization —The localization of the site of a neurologic lesion is deduced by correlating the abnormalities noted in sensation muscle movement and the reflex arc Lesions that involve a peripheral or cranial *sensory*

CHAPTER 60

THE VOLUNTARY NERVOUS SYSTEM CONGENITAL AND HEREDITARY ABNORMALITIES

Anencephaly and Hemicephaly
Porencephaly
Cyclops
Microcephaly
Cerebral and Cerebellar Agenesis
Cranium Bifidum
Congenital Hydrocephalus
Anomalies of the Cord
Spina Bifida
Amaurotic Family Idiocy (Tay Sachs Disease)
Niemann Pick's Disease (p 1134)
Gaucher's Disease (p 1133)
Hand Christian Schuller's Disease (p 1137)
Xanthochromatosis (p 113)
Gargoylism
Tuberous Sclerosis
Multiple General Neurofibromatosis (von *Recklinghausen's Disease*)
Hereditary Spinal (Friedreich's) and Cerebellar (Marie's) Ataxia
Family Periodic Paralysis
Hereditary Chorea (Huntington)
Progressive Lenticular Degeneration (Wilson)
Aplasia Axialis Extracorticalis Congenita (Pelizaeus Merzbacher)
Pseudohypertrophic Muscular Dystrophy (p 2880)
Progressive Muscular Atrophy (p 2884)
Myasthenia Gravis (p 2886)
Myotonia Congenita (Thomsen Disease) (p 2886)
Myotonia Atrophica (p 2888)

The majority of congenital anomalies of brain and cord are mere descriptive entities for which treatment holds no promise Spina bifida cranium bifidum and congenital hydrocephalus however present therapeutic potential in the hands of the expert neurosurgeon

ANENCEPHALY AND HEMICEPHALY

Absence of the entire brain or half of the cerebrum is incompatible with life The child may survive delivery but succumbs shortly thereafter

PORENCEPHALY

In porencephaly there are small or large unilateral or bilateral defects within the cerebral hemispheres The condition may be the result of defective development although occasionally it follows cerebral hemorrhage trauma or postnatal infection The cavity formations produce marked cerebral atrophy and the child may survive for a few years in an asymptomatic phase as a *mental defective* an *epileptic* or a *paralytic*

Operative Procedures

1 Decompressive craniotomy (p 1429)
2 Decompressive laminectomy (p 1434)
3 Ventriculostomy in hydrocephalus (p 2774)
4 Electrocoagulation of the choroid plexuses in hydrocephalus (p 1409)
5 Operative removal of brain and cord tumors (p 2774)
6 Intraspinal drainage and subarachnoid injection therapy (p 1434)
7 *Intracisternal drainage and therapy (p 3783)*
8 Intraventricular drainage and injection therapy (p 3783)
9 Relief of pain by chordotomy
10 Removal of coccyx for coccydynia (p 3073)
11 Sacral caudal and epicaudal anesthesia (p 3972)
12 Removal of prolapsed nucleus pulposus
13 Division of the scalenus anticus for relief of brachial neuralgias
14 Injections of the sphenopalatine ganglion or the gasserian ganglion in facial neuralgias (p 1482)
15 Resection of the posterior root of the fifth nerve for facial neuralgia (p 1482)
16 Drainage of cerebral spinal cord and meningeal abscesses
17 Removal of epidural and subarachnoid clots
18 Division of adhesions of meninges and of ligamentum flavum
19 Local infiltrations of procaine for trigger points'
20 Neurectomy and neurolysis for peripheral nerve disturbances
21 The surgery of the involuntary nervous system (p *1394*)

CHAPTER 69

THE VOLUNTARY NERVOUS SYSTEM: CONGENITAL AND HEREDITARY ABNORMALITIES

Anencephaly and Hemicephaly
Porencephaly
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Xanthochromatosis (p 113[illegible])
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19 Local infiltrations of procaine for 'trigger points
20 Neurectomy and neurolysis for peripheral nerve disturbance
21 The surgery of the involuntary nervous system (p 1394)

CRANIUM BIFIDUM

Cranium bifidum occurs in the infant as a congenital defect in the skull Through this there may be a hernial protrusion of brain (*encephalocele*) or meninges (*meningocele*) The hernial protrusions usually appear in the mid dorsal line they are most common in the occipital region though they may be ventral and extend into the mouth Other associated congenital anomalies include harelip congenital dislocation of the hip club foot and cleft palate



F g 60—Ventr culograms lateral and ante oposterior show ng the hu e dilatation f the ventr cles in hydroc phalus

At first recognition of the anomaly the practitioner insists upon con sultation with the neurosurgeon for *plastic repair* The optimum time for operation is the end of the third or fourth week of life

CONGENITAL HYDROCEPHALUS

Congenital hydrocephalus may be external or internal the latter may be of the obstructive or communicating type and unilateral or bilateral

External Hydrocephalus—External hydrocephalus is more properly re garded as a *subarachnoid accumulation of fluid* In this rare condition the

Reichert, in Ch istopher Textbook of Su gery

CYCLOPS

The cyclops is a monster with a single medially situated eye and orbit The condition results from a defective development of the anterior cerebral vesicle

MICROCEPHALY

In microcephaly there is a defective development of the whole brain with premature ossification of the skull The microcephalic almost invariably is an *idiot* an *imbecile* or an *epileptic* The surgical approach to therapy by trephining the skull is useless

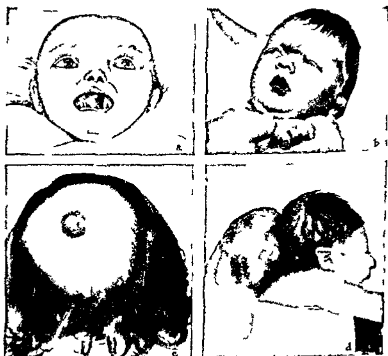


Fig 259—Location of meningoceles *A* Into the mouth through a *cranium bifidum* at the base of the skull *B* Frontal cranium bifidum with meningocele *C* Posterior parietal *cranium bifidum with small meningocele D Occipital cranium bifidum with encephalomeningocele* The nervous tissue was connected with both cerebral hemispheres

CEREBRAL AND CEREBELLAR AGENESIS

Cerebral or *cortical agenesis* which may involve one or both hemispheres produces mental deficiency and cerebral palsies The *cerebellar type of agenesis* is characterized by ataxia and comparatively slight mental defect It may be associated with cerebral agenesis under which circumstance the symptoms of the latter are superimposed Cranial nerve involvements particularly of the facial and ocular nerves may be present *Treatment* is symptomatic

MACROCEPHALY

See *Hydrocephalus* (p 1409)

* Con in Christopher Textbook of Surgery

CRANIUM BIFIDUM

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Fig 260—Ventriculograms lateral and anteroposterior showing the huge dilatation of the ventricles in hydrocephalus

At first recognition of the anomaly the practitioner insists upon consultation with the neurosurgeon for *plastic repair* The optimum time for operation is the end of the third or fourth week of life

CONGENITAL HYDROCEPHALUS

Congenital hydrocephalus may be external or internal the latter may be of the obstructive or communicating type and unilateral or bilateral

External Hydrocephalus—External hydrocephalus is more properly regarded as a *subarachnoid accumulation of fluid* In this rare condition the

brain fills only a portion of the cranium and the remainder of the cavity is taken up with an excess of cerebrospinal fluid Lumbar puncture reveals an abnormal amount of yellow fluid which flows freely contains a high content of protein and reaccumulates with great rapidity No blockage is demonstrable and dye injected by lumbar puncture appears in the ventricles in appreciable quantities within half an hour

Internal Hydrocephalus—Internal hydrocephalus may be unilateral or bilateral In the *communicating types* of the affliction, there is no blockage *in the ventricular system but the defect is in the absorbing channel*

The *obstructive type* of congenital hydrocephalus is usually due to some congenital defect neoplasm or stricture which blocks one or both



Fig 261—An extreme case of hydrocephalus This boy age three years had an almost normal *mentality* *

ventricles Spinal puncture reveals a diminution in the amount of fluid injection of a dye such as neutral phenolphthalein through the fontanelle and into the ventricle fails to produce significant excretion in spinal fluid or urine The ventricular fluid is clear its protein content does not exceed 15 mg and its basal pressure exceeds 50 mm of water

Clinical Manifestations—Congenital hydrocephalus is usually recognized at birth through the enlargement of the head During attempts at delivery it may cause *dystocia* or death of the child The head is large out of proportion to the body the scalp is thin, the veins are prominent the face appears small and triangular the eye sockets are widely separated the temples overhang the sutures are wide the fontanelles are tense and pulsating the eyeballs converge the sclerae are visible the child is feeble

* Dandy in Lewis "Practice of Surgery W F Prior Co Publisher

and convulsions occur often Optic atrophy and ocular palsies are observed The child has difficulty in feeding and usually succumbs within a short time Should it survive its development is delayed changes may be observed in the muscles or deep reflexes and intercurrent infection is almost inevitable The skull which has a normal average circumference of 35 to 40 cm at birth may measure 60 to 100 cm On rare occasions positive serological tests for syphilis are demonstrable

Treatment—The hydrocephalic child is referred immediately to the neurosurgeon The external type recognized by the excess of xanthochromic cerebrospinal fluid of high protein content is not responsive to treatment However exploration may be justified on the chance that the condition is a subdural hematoma amenable to surgical therapy

Internal hydrocephalus of the communicating type may be managed by *bipolar endoscopic coagulation of the choroid plexus* This technic requires the services of an experienced expert and should not be attempted unless the mental and physical status of the infant approaches normal Perpetuation of life in an idiotic or hopelessly paralyzed child is not encouraged unless the parents are insistent Many surgeons object to the endoscopic approach since coagulation is effected over the basal ganglia they prefer open operation after application of a plaster cast to the head

In obstructive hydrocephalus an attempt may be made to provide drainage by *ventriculostomy* of the third ventricle Palliative procedures consist of frequent *lumbar taps* in communicating hydrocephalus and repeated *ventricular aspirations* in the obstructive types Congenital syphilitics are given intensive antiluetic therapy (p 2787)

ANOMALIES OF THE CORD

Anomalies of the spinal cord are of rare occurrence They include partial or complete absence *diplomyelia* (splitting of the cord) and *heterotopias* in which the gray matter is displaced in the white substance

None of these disturbances hold any therapeutic possibility and their presence usually is not revealed until postmortem examination

SPINA BIFIDA

Spina bifida (rachischisis) is a developmental defect of the spinal column In the *occulta* variety the bony defect is covered by skin it may be asymptomatic or cause disturbances that are observed only in later life

Spina Bifida Manifesta—Spina bifida manifesta is noted upon the birth of the child as a median line tumor whose usual location is the lower lumbar region Its hernial sac may contain meninges (*meningocele*) or cord tissue (*myelomeningocele*) and the associated neurological manifestations may be mild or severe Other coexistent congenital malformations include hydrocephalus cleft palate harelip or anomalies of the kidney

Clinical Manifestations—The clinical manifestations of spina bifida may be tegumentary or neurologic Often the condition is suspected by the appearance of a dimple a local area of pigmentation or hypertrichosis in the median line overlying the defect The neurological phenomena include incontinence of urine or feces weakness or paralysis of the legs and the appearance of trophic ulcers on the extremities Roentgenograms reveal the defective development of the arches

Treatment —Asymptomatic spina bifida requires no therapy unless the overlying skin is thin and there is danger of rupture and contamination With mild or severe neurologic symptoms operative interference is justified as soon as the infant s condition is stabilized Occasionally exploration reveals divisible bands that have compressed the cauda or a lipoma which can be excised Otherwise the procedure consists of repair of the sac and skin closure

AMAUROTIC FAMILY IDIOCY (TAY SACHS DISEASE)

Amaurotic family idiocy is a congenital malformation in which there is a degeneration of the ganglion cells of the central nervous system revealed by examination of the retina It appears almost exclusively in Jewish children and is fatal within two years or a little more An adolescent

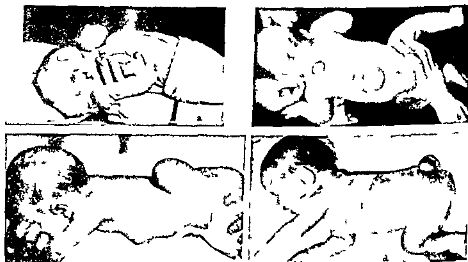


Fig 262 —*A* Cervical spina bifida with meningocele *B* Dorsal spina bifida with simple meningocele and lumbar spina bifida with myelomeningocele *C* Lumbar spina bifida with myelomeningocele *D* Sacral spina bifida with meningocele

form occurs in non Jewish children at the ages of seven to eight The pathologic changes suggest that the syndrome is related to Niemann Pick s disease (p 1134)

In its usual form the *clinical manifestations* of amaurotic family idiocy become evident about the fourth or sixth month of life The child which had been developing normally seems no longer able to recognize its mother or its bottle It has difficulty in holding up its head the limbs appear weak and paralyzed and there is sensory hyperacuity as revealed by an excessive response to noise Additional manifestations include nystagmus strabismus and convulsions

The *diagnosis* is established by the ophthalmoscopic finding of the pathognomonic cherry red spot with a surrounding white halo With the progress of time the child deteriorates until it is a vegetating idiot Death usually occurs within the year from intercurrent infection or malnutrition

* Cone in Christopher Textbook of Surgery

NIEMANN PICK S DISEASE

See p 1134

GAUCHER S DISEASE

See p 1133

HAND CHRISTIAN SCHULLER S DISEASE

See p 1137

XANTHOCHROMATOSIS

See p 1135

GARGOYLISM

Gargoylism resembles amaurotic family idiocy (p 1412) except that optic atrophy is not present After the first few months of life the child

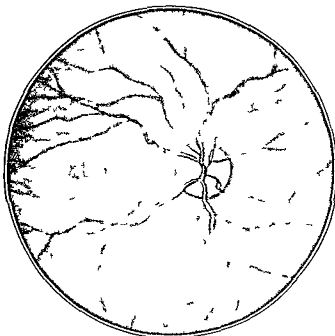


Fig 263—Cherry red spot and optic atrophy in amaurotic family idiocy and Niemann Pick disease

appears to be abnormal Vision is defective the arms and legs are short there is a kyphosis the head is large the lips are coarse the eyebrows are bushy the neck is short the abdomen is large and examination reveals an umbilical hernia and enlargements of liver and spleen The child resembles an achondroplastic dwarf and is obviously defective mentally It may survive for many years despite retardation

TUBEROUS SCLEROSIS

In its fully developed form tuberous sclerosis is characterized by progressive mental deterioration idiocy epilepsy and tumors of the skin and

Courtesy of Drs I Goldstein and D Wexler Arch Ophthal May 1931

viscera The condition may be mild in its partially developed forms or it may be thoroughly incapacitating



Fig 264—Sebaceous adenoma in tuberous sclerosis



Fig 265—Multiple general neurofibromatosis

The characteristic *tegumentary manifestations* include pigmentations and tumors of the skin cafe au lait spots fibromas nevi and *sebaceous*

adenomas (p 3148) over nose and cheeks The *internal visceral lesions* include rhabdomyomas of the heart and fibromas of the intestines and kidneys In rare instances a *phacoma* of the retina is observed The *cerebral changes* consist of nodules of glial tissue which may become transformed into gliomatous tumors resembling glioblastoma multiforme

The *neurologic manifestations* are most often observed in early childhood The infant appears sullen and retarded older children suffer from conduct disorders epileptic convulsions and idiocy The coincidental presence of the dermatological phenomena suggests the diagnosis

Unfortunately the recognition of tuberous sclerosis has only academic importance since there is no available method of therapy that has any promise of benefit Children who are badly handicapped should be institutionalized

MULTIPLE GENERAL NEUROFIBROMATOSIS (VON RECKLINGHAUSEN S DISEASE)

Von Recklinghausen s disease is a congenital and hereditary disturbance characterized by the growth of numerous *fibromas* or *neuromas* The tumors most often grow on sensory nerves of the skin and are accompanied by tegumentary changes in the nature of *pigmented nevi* and *diffuse pigmentations* suggestive of tuberous sclerosis (p 1413)

Multiple neurofibromatosis may present minimal signs or a wide variety of significant clinical manifestations Painful and tender nodules (*tubercula dolorosa*) may require operative removal In *elephantiasis neuromatosa* the tumors are associated with diffuse thickenings of the skin subcutaneous tissues and bones Tumors may grow in awkward positions such as within the spinal canal intracranially or in the cerebellopontine area (p 1426) Very occasionally neoplasms of long duration suddenly develop malignant characteristics

See *Neoplasms of the Cord* (p 1430) *Neoplasms of the Brain* (p 1419)

The *treatment* of von Recklinghausen s disease involves local excision of tumors which are large painful invasive or productive of compression phenomena relative to nearby structures

HEREDITARY SPINAL (FRIEDREICH S) AND CEREBELLAR (MARIE S) ATAXIA

Hereditary ataxia is a chronic familial and progressive degenerative disorder It usually affects several children in a family and is first observed between the ages of six and fifteen The pathological lesion consists of degenerative changes in the posterior and lateral columns pyramidal and spinocerebellar tracts and cerebellum

Friedreich s Spinal Ataxia—The *clinical manifestations* of Friedreich s disease are unmistakable The presenting findings are ataxia weakness of the legs pes cavus equinovarus (clubfoot) scoliosis manus cava nystagmus tremor scanning speech and absent reflexes The dorsum of the foot is high the arch is deeply hollowed and the great toe is hyperextended in the position of a permanent Babinski Sphincteric control is maintained and there are no disturbances in sensation The ataxia being of the cerebellar type is present equally with eyes opened and shut Ocular mani

festations include retinitis pigmentosa juvenile cataracts ocular palsies and atrophies

Marie s Cerebellar Ataxia —The Marie form of cerebellar ataxia represents a variant of Friedreich s disease in which spinal manifestations are minimal

Diagnosis and Treatment —The diagnosis of spinal or cerebellar ataxia is an academic discipline as there is no present method of therapy *Juvenile tabes* (p 1465) is differentiated by the spinal fluid findings *multiple sclerosis* (p 1504) occurs in older patients

FAMILY PERIODIC PARALYSIS

Family periodic paralysis is a rare disease of the nervous system in which there are attacks of paralysis of the muscles of trunk and extremi



Fig °66 —Typical foot in Friedreich s ataxia

ties The individual episodes may last for twenty four hours to several days and occur at intervals of a few days to many months The paralysis usually comes on at night there are no sensory disturbances the cranial nerves are rarely involved and the paralyzed muscles cannot be stimulated electrically

Hypophosphatemia —The onset of the disturbance is associated with an acute fall in the potassium content of the blood The normal level of 18 to 23 mg per 100 cc may be reduced to 10 mg or less There is a suggestion that the hypophosphatemia is precipitated by the ingestion of a meal that is rich in carbohydrate (p 672)

Treatment —The prevention of attacks of periodic paralysis is accomplished by avoiding high carbohydrate meals and administering 4 to 8 cc of *25 per cent potassium chloride* two to four times daily as needed Ac

* Neurological Service of the Montefiore Hospital

tive therapy consists of the use of hourly doses of 8 cc of the 25 per cent potassium chloride solution until symptoms disappear In extremely severe paralyses involving the respiratory muscles 50 cc of a 2 per cent *potassium chloride solution* may be injected intravenously followed by oral doses in the manner previously described Injections of *adrenal cortical extract* (p 1267) have been suggested through the resemblance of the condition to the acute crises of the Addisonian syndrome (p 1271)

HEREDITARY CHOREA (HUNTINGTON)

Huntington s chorea is a rare hereditary and familial affection in which there is a *chronic progressive chorea* accompanied by *mental deterioration* The disease begins in the fourth decade and is transmitted in direct line through several generations The cause is unknown but the pathologic



Fig 67 —Wilson s disease showing associated dental abnormality malocclusion and gingivitis

process which involves the whole brain consists of atrophy of fibers and cells with degeneration of ganglia

Clinical Manifestations —Huntington s chorea begins gradually and first affects only limited parts of the body The *choreic movements* are irregular purposeless and disorderly with facial grimacing a bizarre gait clownish movements clumsy gesticulations dysarthria smacking of the tongue and lips and sudden jerking of the legs The movements are exaggerated by effort tension and emotional stimuli but they disappear at night The *mental deterioration* is initiated by irritability aprosexia impairment of memory apathy suicidal tendencies and progressive impairment to the point of imbecility or total dementia

Course and Treatment —Huntington s chorea runs a progressively unfavorable course and usually requires institutionalization Some symptomatic

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CHAPTER 70

THE VOLUNTARY NERVOUS SYSTEM NEOPLASMS

Tumors of the Brain
Neoplasms of the Spinal Cord
Neoplasms of the Peripheral Nerves

NEOPLASTIC disease of the voluntary nervous system may involve the brain cord or peripheral nerves Secondary metastases from distant malignancies are of lesser importance to the practitioner than primary tumors Ad vances in neurosurgical technic have greatly lessened the risks of craniot omy and *laminectomy* These procedures should be undertaken whenever there is hope of partial or complete removal of a neoplasm

TUMORS OF THE BRAIN

Tumors of the brain are encountered with great rarity in private prac tice Nevertheless the physician is required to be on the alert for cerebral neoplasms Institutional experiences reveal unsuspected instances in pa tients with psychiatric changes of sufficient moment to have warranted commitment to mental hospitals

The nature and incidence of brain tumors are best illustrated by pres entation of the chart prepared at the clinic of the late Harvey Cushing (Table 92) The record probably reveals an unusually high percentage of pituitary adenomas through Cushing s particular interest and inordi nate skill in the management of these lesions

The Cush g tables reveal that the brain tumors that occur with appreciable frequency a e *gl omas pituitary adenomas* *n n ng omas acoustic neurinomas* and *craniopharyngiomas* As the pitu tary adenomas (p 11 5) and the cran opl aryngiomas (p 117) are elsewhe e de s ribed the present discussion is l mited to a consideration of gliomas meningiomas and acoustic neu inomas

Gl om —The gl oma is the most important and most common brain tumor It is nearly always single often infilt ates the brain and may sp ead by way of cerebrosp nal flu d path ways It s usually su rou ded by an area of necrosis and is occasionally the s te of *hemorrhage* and *cystic degeneration* Gliomas may be small or so la ge as to infiltrate a hem isphere

Astrocytomas—Of the clas fiabl gliomas the commonest are astrocytomas These are slow growing c pable of removal and somewhat rad osensitive Generally they occur in the *ce ebr l hemispheres* of ad lts and the *ce ebella lobes* of children They are som what more c cumscribed than oth r gl omas they oft n cal fy and frequ ntly become necrotic or cystic The tumo has compa atively few vessels but many glial fibers and protoplasmic processes (*fibrilla y and protoplasm c ast ocytomas*) Ce ebellar ast ocyt mas in ch ldhood a e pa t cula ly menable to surgical removal the cereb al tum r how ver can seld m be com pletely removed

Gl oblast m s and M d lloblastomas—The gl obla toma multiforme (*gliosarcoma*) grows rapidly and mainly affects adults of m ddle age It gen ally involves the cer b al hem ispheres and is subje t to cystic or necrot c d generat on *Medulloblastomas* are rapid grow ing but rad osensitive growths that g ne ally invade the cerebellum in children

M g m (E dothel om Me goth l om Ar h o d l o M geal F b oblastoma) —Men ngiomas a e the commonest conn t ve t ssue tumors of the bra n They occur along

relief is afforded by the administration of bromides barbiturates or hyoscine

PROGRESSIVE LENTICULAR DEGENERATION (WILSON)

Wilson s disease is a rare familial condition which affects young people and is associated with a *hobnail cirrhosis of the liver* (p 1969) The afflicted show bilateral rhythmic *athetoid movements* of hands arms legs and face The emotional disturbance is reflected by a *perpetual grin* and *spasmodic laughter* The limbs are spastic and eventually develop contractures, reflexes are active but there is no Babinski sign and no actual weakness of muscles or loss of sensation abdominal reflexes are preserved speech is dysarthric and there is difficulty in swallowing

The disease progresses inexorably and death occurs within a few years

APLASIA AXIALIS EXTRACORTICALIS CONGENITA (PELIZAEUS-MERZBACHER)

Pelizaeus Merzbacher s disease is a rare congenital and familial abnormality which begins in infancy The chief clinical features are slowness of speech fixation of facial expression nystagmus ataxia spasticity of the legs and mental deficiency The lesion is an atrophy of the white matter of the brain The course is chronic and uninfluenced by treatment

may also occur *spongioblastomas* which sometimes involve the pons or optic chiasm in children *astroblastomas* *oligodendrogliomas* *ependymomas* which arise in or near the ventricles and tend to calcify *papillomas* of the choroid *sarcomas* which are usually secondary *angiomas* *dermoids* *cholesteatomas* which occur at the base of the brain *osteomas* *osteosarcomas* *enchondromas* *chondrosarcomas* *lipomas* and *metastatic carcinomas*

Clinical Manifestations—The clinical manifestations of the brain tumor may be those of a general increase in *intracranial pressure* or they may be due to *local disturbances* of involved neighboring structures Occasionally *acute degeneration* or *bleeding* gives the symptoms of a non neoplastic cerebral vascular accident (p 1430)

Increased Intracranial Pressure—The generic syndrome of increased intracranial pressure includes headache vomiting dizziness papilledema



Fig 268—Calcified glioma

convulsions psychic or mental disturbances and alterations in pulse and respiratory rates

HEADACHE AND PERCUSSION TENDERNESS—Headache is often the first sign of brain tumor it may be violent and persistent or relatively mild and paroxysmal it may be diffuse or local and under the latter circumstance it is often accompanied by percussion tenderness of the skull *Localized spontaneous cephalalgia* with corresponding percussion tenderness has some slight diagnostic value but may be most misleading

NAUSEA AND VOMITING—Vomiting is almost universally present in tumors of the posterior fossa (p 1493) It has no relation to meals and may or may not be preceded by nausea Often it is *projectile* probably as the result of irritation of the fourth ventricle or the basal ganglia

DIZZINESS—Dizziness is an important complaint in *infratentorial neoplasms* such as tumors of cerebellum cerebellopontine angle medulla and

the courses of the *meningeal vessels* and the *superior longitudinal sinus* as hard, slow growing vascular neoplasms which invade the dura and produce erosion and thinning or hyperostosis of the overlying skull The tumors grow on the surface of the brain but they do not infiltrate When they become calcified they form *brain sand* and are known as *psammomas* Certain meningiomas tend to recur even if completely removed

TABLE 92 —THE NATURE AND INCIDENCE OF BRAIN TUMORS

(From the Clinic of the late Harvey Cushing)

		Number	Percentage
Gliomas		860	43 0
Pituitary Adenomas		3[illegible]	17 9
Chromophobe	26[illegible]		
Chromophile	8		
Mixed	23		
Meningiomas		26[illegible]	13 2
Neurinomas (acoustic)		170	8 5
Congenital Tumors		111	5 6
Craniopharyngiomas	91		
Cholesteatomas and Dermoids	14		
Chordomas and Teratomas	6		
Metastatic and Invasive Tumors		[illegible]9	3 9
Granulomatous Tumors		47	[illegible] 3
Tuberculomas	3[illegible]		
Syphilomas	15		
Blood vessel Tumors		41	[illegible] 0
Sarcomas (primary)		1[illegible]	0 6
Papillomas (choroid plexus)		11	0 5
Miscellaneous and unclassified		50	[illegible] 5
Total		[illegible]001	100 0

* *Verified gliomas were as follows*

Unclassified			211
Verified by cystic fluid alone		61	
Excluded because differential diagnostic impossible		[illegible]	
Gliomas of optic chiasm		23	
Gliomas of pons and midbrain		2[illegible]	
Atypical gliomas		3[illegible]	
Transitional forms		6	
Classified			649
Neuro-epithelioma		2	
Medulloblastoma		87	
Pinealoma		9	
Ependymoma		23	
Glioblastoma multiforme		196	
Spongioblastoma polare		18	
Oligodendroglioma		27	
Astroblastoma		31	
Astrocytoma		250	
Fibrillary	152		
Protoplasmic	98		
Ganglioneuroma		3	
Total			860

Acoustic Neuromas (Neurinomas or Neurofibromas (Perineural Fibroblastomas))—Acoustic neuromas neurinomas or neurofibromas are encapsulated tumors growing on the eighth nerve They are slow growing and are comparatively easily removed They may be part of a more general neurofibromatosis such as von Recklinghausen's Disease (p 1415)

Miscellaneous Tumors—Besides the common tumors of the Cushing classification there

sciousness suggesting epilepsy facial tics and tonic spasms may be herald manifestations of an intracranial neoplasm The sudden onset of motor irritation in a previously healthy adult is a sufficient indication for a complete survey by the specialist even to the point of exploratory craniotomy

VISCERAL PHENOMENA —With increasing intracranial pressure the *pulse rate* becomes slower and *respirations* may be depressed accelerated or of the Cheyne Stokes type *Hiccough is a rare symptom but alterations in temperature* may accompany alterations in pulse and respiratory rates Tumors of the floors of the third and fourth ventricles may produce *glycosuria diabetes insipidus adiposis* and other pituitary manifestations (p 1159)

Localization of Brain Tumors —The localization of the brain tumor is specialist province The diagnostic investigation is approached by clinical examination roentgenography ventriculography encephalography spinal fluid examination electroencephalography and exploratory craniotomy



Fig 2 0—Hyperostosis from meningioma of *tuberculum sellae*

Since the operability of the brain tumor is often dependent upon early diagnosis the practitioner is required to become familiar with localizing symptoms so that the patient may be referred promptly to the neurosurgeon for more elaborate investigation and therapy "Reaction patterns are independent of the nature of the brain tumor whether neoplastic inflammatory or vascular They *may* be superimposed on the manifestations of *increased intracranial pressure* or they may exist independently

Meningeal Reaction Pattern—Inflammations and hemorrhages involving the meninges are easily recognized through cytologic serologic and chemical changes in the *cerebrospinal fluid* (p 2734) Unfortunately for diagnostic purposes *meningiomas* which usually arise from the *arachnoid* cells seldom produce such easily recognized manifestations Their presence is suggested by radiographs of the skull which reveal increased *local arterialization* and changes in the density of the overlying bone and by *focal manifestations* which result from pressure on the underlying brain tissue Only late in the course does the meningioma attain sufficient size to cause signs of *increased intracranial pressure* (p 14 1)

Lateral Ventricle Block Reaction Pattern—The paired lateral ventricles lie deep in the brain Their *floor* is made up of thalamic bodies the *lateral walls* are adjacent to the in

pons *Tumors of the fourth ventricle* (p 1424) are characterized by the onset of dizziness with change in position

PAPILLEDEMA —*Choked disk* (papilledema) is the *most characteristic single sign* of brain tumor It occurs most commonly when there is blockage of the aqueduct of *Sylvius* with resultant *internal hydrocephalus* (p 1410) Thus it is almost *constant in* tumors of pineal quadrigeminal plates and posterior fossa it is *also seen* in neoplasms of occipital temporal parietal and frontal regions Unless relieved the choked disk leads to field defects optic atrophy impairment of vision and eventual blindness Tumors that press *directly* on the optic nerve chiasm and tracts may produce as a first sign an *optic atrophy*

PSYCHIC AND EMOTIONAL CHANGES —Psychic and emotional changes are of frequent occurrence Drowsiness yawning clouded consciousness stu

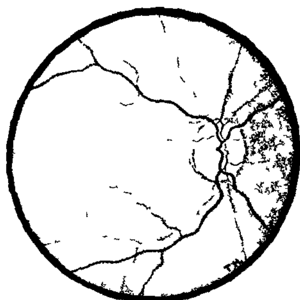


Fig 269 —Simple optic atrophy

por loss of memory personality changes moral defects delusions hallucinations delirium mania facetiousness puerility sexual aberrations and insomnia or somnolence may be encountered Tumors of the third ventricle and *pineal* often cause hypersomnia personality and sexual aberrations distinguish frontal lobe and thalamic *tumors* *See* p *1424*

Even highly trained psychiatric staffs have the experience of discovering at postmortem an unsuspected brain tumor in a patient who had been confined as a psychopath This diagnostic error can be avoided only by *suspecting* an intracranial growth in each characterological or psychiatric manifestation of recent or sudden onset Often the diagnosis is clarified only by *exploratory craniotomy*

CONVULSIONS —Convulsions occur most commonly with lesions in or near the motor cortex and the temporosphenoidal lobes *The convulsions may* be *generalized* or *focal* (p 1519) An attack of petit mal a loss of con

Gifford Textbook of Ophthalmology

Block of the fourth ventricle may occur from an intrinsic lesion such as an *ependymoma* or a *polar spongioblastoma* The former is not infrequent in adults while the latter occurs in childhood or adolescence Blockage of the fourth ventricle may be intermittent with acute paroxysms of *severe headache nausea vomiting* and *alterations in pulse and respiratory rates* The attacks often occur with changes in head position

Anterior Fossa Reaction Pattern—The anterior fossa contains *frontal lobes* and the *first cranial nerves* The *olfactory nerve sheath* is a not infrequent site for the origin of a *meningioma* The presenting symptom is *anosmia* followed by the signs of *increased intracranial pressure* (p 1421) Radiographs of the skull reveal thickening of the bone and increased vascularization

In the *Foster Kennedy* syndrome the anosmia is associated with ipsilateral optic atrophy and contralateral papilledema

Frontal Lobe Reaction Pattern—The frontal lobes are the great association and integration areas for all functions of the nervous system Destruction produces changes in *character* or *personality* manifested by euphoria purposeless jocosity incontinence of urine unethical conduct abnormal sexual behavior anxiety regressions nervousness inattention yawning fear distractibility poor judgment and impairment of memory or intellect *Ab*

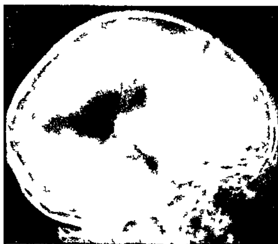


Fig —Rounded defect in third ventricle frontal area of third ventricle

normal movements (grasping and groping) inability to carry out skilled acts (*apraxia*) and pyramidal tract dysfunctions occur when the posterior portion of the lobe is destroyed Additionally there may be adrenergic hypothalamic functions increased contralateral cerebellar signs and symptoms and aphasia

Lesions in the *prerolandic area* involving the motor pathways produce focal irritative or convulsive symptoms (*Jacksonian epilepsy*) Later they lead to contralateral hemiplegia without sensory signs until the *postrolandic area* in the parietal lobe becomes involved Left sided tumors in right handed patients cause motor aphasia

Middle Fossa Reaction Pattern—The middle fossa contains *parietal occipital* and *temporal lobes sella turcica pituitary gland optic chiasm optic tracts* and *third fourth sixth* and *fifth cranial nerves* Pituitary lesions cause erosion of the clinoid processes of the sella and enlargement deepening and thinning of the pituitary fossa Meningiomas which usually arise along the vertex or parietal area result in thickening of the inner layer of the skull and increased vascularization

Parietal Lobe Reaction Pattern—The parietal lobe is the great sensory center of the brain Disturbances produce impairment or absence of one or more *cortical sensory modalities* over the *contralateral half of the body* This results in *defective sense of body position* with *astereognosis apraxia incoordination and slight weakness* of the affected arm and leg

Peet in Christopher Textbook of Surgery

ternal capsules and the *roof* impinges upon the multiple association fibers between the various centers and the cerebral hemispheres the *temporal horns* are in close relationship to the cortex of temporal lobes and visual pathways

Block of the lateral ventricle is usually *unilateral* and due to a slow growing malignant *ependymoma* The blockage produces manifestations of *internal hydrocephalus* and localizing symptoms which depend upon the pressure upon impinging structures The internal hydrocephalus is recognized by headache nausea vomiting changes in pulse and respiratory rates somnolence and coma Downward invasion adds the *thalamic reaction pattern* (p 1426) *lateral invasion* produces disturbances of the internal capsule with *contralateral hemiparesis* and/or *hemisensory defects and hemianopsia*

An early sign of ventricular displacement is a *shifting of the pineal gland* as seen on lateral or anteroposterior radiographs of the skull when the gland contains enough calcium to cast a shadow More accurate localization is provided by *ventriculography*

Third Ventricle Block Reaction Pattern—The unpaired third ventricle lies between the basal ganglia Its *anterior wall* is adjacent to hypothalamus optic chiasm and pituitary gland the *posterior wall* is in close relationship to pineal gland and secondary nuclei for hearing and vision (corpora quadrigemina) The third ventricle joins the *lateral ventricles*



Fig 971—Ventricular displacement from cystic ependymona The cyst and ventricular system were injected with oxygen independently

by the short foramina of Monro and the *fourth ventricle* by a long narrow iter the aqueduct of Sylvius

Block of the third ventricle is rare and may be caused by an *astrocytoma* or a *congenital colloid cyst* A lesion of the third ventricle which occludes the *foramen of Monro* produces localized *internal hydrocephalus* of the lateral ventricle as previously described (p 1410)

Occlusion of the iter dilates both lateral ventricles as well as the third ventricle *Posterior dilatation* of the third ventricle displaces posteriorly the pineal gland and through pressure on the corpora quadrigemina causes bilateral impairment of the upward movement of the eyes *Lateral anterior and downward dilatations* give rise to hypothalamic reaction pictures (p 1160) In any event involvements of optic chiasm and/or optic nerves result in bilateral papilledema

Fourth Ventricle Block Reaction Pattern—The fourth ventricle begins at the level of the pons and is located on the roof of pons and medulla The roof of the fourth ventricle itself is thin and lies immediately beneath the mesial portions of both cerebellar hemispheres and the vermis

Peet, in Christopher Textbook of Surgery

are resultant difficulties in articulation swallowing and the rate and regularity of respiration and heart

Pontine Reaction Pattern—Pontine reaction patterns are characterized by *crossed paralyses* which consist of a disturbance of the *ipsilateral cranial nerves* (V VI and VII) and the *contralateral arms and/or legs* papilledema appears late

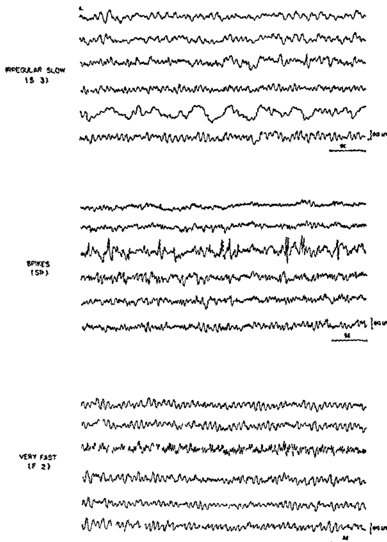


Fig 275—Types of electro-encephalographic focus that are very reliable indicators of local d organic brain damage Time and voltage calibrations at the lower right in each case

Midbrain Reaction Pattern—Midbrain reaction patterns are rarely neoplastic The syndrom of *Wernicke's disease* which is probably a vitamin deficiency is elsewhere described (p 1502) A characteristic syndrome (*Weber*) includes ipsilateral paralysis of III with contralateral hemiplegia

Gibbs in Manual of Military Neuropsychiatry by Solomon and Yakovlev

The patient confuses right and left halves of the body Fairly frequently there is a *homonymous quadrant visual defect* or *hemianopsia* The reflexes on the affected side are variable and may be reduced absent or hyperactive

Temporal Lobe Reaction Pattern—The temporal lobe is the great reception center for hearing equilibration taste smell vision and speech *Lesions* are characterized by *aphasia* and *defects in the visual field* which are more pronounced when the dominant lobe is affected (left temporal lobe in right handed person) The visual defect is either a *quadrantic or a hemianopic homonymous field defect* apparent on tangent screen examination (p 1643) There may be dream states psychic fits and auditory hallucinations as in the psychoses A *contralateral hemiplegia* with pyramidal weakness may be most marked in the lower face tongue and arms *Uncinate fits* are infrequent but characteristic

Occipital Lobe Reaction Pattern—The occipital lobe is often silent involvement may produce *word blindness* and *homonymous hemianopsia* on tangent screen examination

Pituitary Gland Reaction Pattern—See *Basophile Adenoma* (p 1175) *Cushing's Syndrome* (p 1159) *Acidophile Adenoma* (p 1175) *Pituitary Dwarfism* (p 1161) *Gigantism* (p 1155) *Acromegaly* (p 1156) *Adiposogenital Syndrome* (p 1166) *Simmonds Disease* (p 1169)

Hypothalamic Reaction Pattern—See *Diabetes Inspidius* (p 1180) *Adiposogenital Syndrome* (p 1166)

Pineal Gland Reaction Pattern—See *Macrogenitosomia Praecox* (p 1185) *Cushing's Syndrome* (p 1159)

Thalamic Reaction Pattern—The thalamic reaction pattern *is* characterized by burning agonizing *intractable pain* over the contralateral half of the body The pain is continuous with acute exacerbations that are initiated by stroking cold emotional stress or strain There may be *ataxia* of the affected arm and leg with associated *abnormal posturing* The hand is cupped with the fingers extended the foot has an accentuation of the arch and the patient walks on the toes There may be contralateral pyramidal tract involvement with *hyperactive* and *pathologic reflexes*

Posterior Fossa Reaction Pattern—The posterior fossa contains brain stem cerebellum basal cisterns and all except the first four cranial nerves The most frequent sites for expanding lesions are cerebellum and cerebellopontine angles

Cerebellar Reaction Patterns—Involvement of a single cerebellar lobe rarely gives symptoms until there has been produced a block of the fourth ventricle the basal cisterns or involvement of the IX XI or XII cranial nerves Early cerebellar symptoms are ipsilateral *incoordination* of the arms and legs unilateral or bilateral *ataxia* with the eyes open or shut *nystagmus* which is slower and of greater amplitude looking toward the side of the lesion and of quicker and lesser amplitude looking contralaterally and slight to moderate rigidity of the neck *Increased intracranial pressure* adds headache nausea, vomiting and papilledema When the *cranial nerves* become involved additional symptoms include corneal areflexia weakness of the tongue palate pharyngeal laryngeal and facial muscles alterations in voice dysphagia, and impairment of facial sensation The reflexes are usually depressed or absent All symptoms and signs are ipsilateral

Bilateral cerebellar involvement is rarely neoplastic and almost always of degenerative origin

Cerebellopontine Angle Reaction Pattern—A common brain tumor is the *acoustic neuroma* which arises from the sheaths of facial or auditory nerves An early manifestation is *unilateral tinnitus* with gradual and progressive *ipsilateral impairment in hearing* there may or may not be attacks of *vertigo* As the neoplasm grows the ipsilateral *face muscle* becomes weakened and then paralyzed in a peripheral manner Examinations reveal *unilateral nerve deafness* with reduction in all responses to *caloric stimulation* (p 2018) A radiograph may show erosions of the internal auditory meatus *Nystagmus* on lateral gaze occurs regularly and early

Later there is reduction in the *pain and temperature senses* of the face especially about the nose and cheeks the *corneal reflex* is absent on the affected side *incoordination* and *ataxia* appear in the ipsilateral arm and leg Finally there are manifestations of *increased intracranial pressure* with nausea, vomiting papilledema and unilateral or bilateral palsy of the sixth nerve Dysarthria, dysphagia and dysphonia suggest involvements of IX and X cranial nerves

Medullary Reaction Pattern—Tumors of the medulla oblongata produce *bilateral signs of pyramidal tract involvement* together with affections of the lowest *cranial nerves* There

are resultant difficulties in articulation swallowing and the rate and regularity of respiration and heart

Pontine Reaction Pattern —Pontine reaction patterns are characterized by "crossed par alyses which consist of a disturbance of the *ipsilateral cranial nerves* (V VI and VII) and the *contralateral arms and/or legs* papilledema appears late

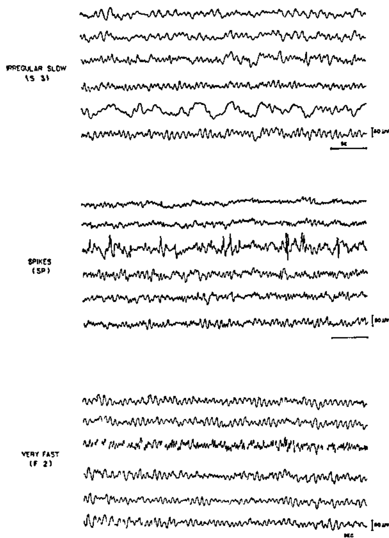


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* From *Manual of Military Neuropsychiatry* by Solomon and Yakovlev

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Cerebrum

With definitive disturbance of the cerebrum the practitioner is required to hold consultation with the neurologist psychiatrist and neurosurgeon The problems involved are usually of serious import and the patient is entitled to a specialist opinion

CAUSE	DIAGNOSTIC FEATURES
Congenital and Hereditary Abnormalities	Anencephaly hemicephaly porencephaly cyclops microcephaly cerebral and cerebellar agenesis macrocephaly cranium bifidum and congenital hydrocephalus (p 1409) Obvious defects noted at birth or immediately thereafter
Idiopathic Psychoses	Schizophrenia manic-depressive insanity involutional psychoses and paranoia (p 1373) Normal clinical and laboratory findings
Symptomatic Psychoses	See p 1374 Note presence of definitive etiologic mechanism in history physical examination neurologic status or laboratory data Remember that other neuropathies may coexist.
Neoplasms	Evidences of increased intracranial pressure (p 1421) cranial nerve palsies papilledema headache nausea and vomiting Refer to specialist for electroencephalogram x ray and exploration
Epilepsy	History of convulsive episodes Characteristic encephalogram. Therapeutic response to phenobarbital and dilantin (p 1517)
Hysteria	With approximate but inaccurate simulation of organic neuropathy (p 1353) Negative laboratory findings
Disturbances of Cerebral Hemodynamics	Temporary alterations due to cerebral anemia hyperemia angiospasm or edema (p 706)
Cerebral Arteriosclerosis	Diffuse disturbances associated with other evidences of vascular disease particularly thickening of peripheral vessels narrowing of retinal artery hypertension and nephropathy (p 2362)
Cerebral Vascular Accidents	Acute and ingravescent disturbances resulting from hemorrhage (apoplexy) thrombosis embolization, aneurysmal dilatation and infection of venous sinuses (p 1446) Cautiously obtain spinal fluid and note evidences of bleeding xanthochromia or increased pressure (p 3734)
Trauma to Skull	Cerebral concussion contusion, laceration or edema may accompany or follow open or closed intracranial injury with or without fracture (p 1450) Look for evidences of cranial nerve involvement and changes in fundus (p 1545) Cautiously obtain spinal fluid (p 3781) Radiography imperative

CONTINUED

Meningitides	By extension from skull fracture and nose or ear infections Secondary to bacteremia and generalized infections as in meningococcus meningitis tuberculosis syphilis and pneu mococcic invasions Positive findings in cerebrospinal fluid (p 1462)
Nonsuppurative Encephalitides	Particularly in virus diseases (p 449) syph ilis (p 1468) tuberculosis (p 1462) rheu matic fever (p 186) and trypanosomiasis (p 531) Positive spinal fluid findings (p 3734)
Suppurative Encephalitides (Brain Abscess)	May occur by continuity from skull fracture or ear and nose infections (p 2128) M ta static in pulmonary suppuration (p 1469) and amebiasis (p 526)
Encephalopathies	Toxic disturbances of cerebrum usually a o-ciated with avitaminoses alcoholi m and poisonings or idiosyncrasy to arsenic lead or mercury
Migraine	Idiopathic recurrent headache on familial and constitutional basis Often relieved by er gotamine tartrate (p 1507)
Cranial Mono or Polyneuropathies	Evidences of cranial nerve paralyses with manifestations of disturbances in the brain in its entirety Note motor or sensory defects or derangements in special sensations such as vision, hearing smell or taste

Diagnosis —The diagnosis of brain tumor is the province of the *special ist* The practitioner cannot be expected to carry the investigation beyond the stage of suspicion He notes the symptoms and physical signs seeks evidences of generalized disease particularly syphilis and tuberculosis per forms a blood serologic test for syphilis obtains the cerebrospinal fluid (p 3734) and determines the cell count and the protein and sugar concentra tions He requests tests for syphilis and the colloidal gold reaction Lym phocytic fluids are examined for tubercle bacilli and injected into guinea pigs The practitioner is warned that lumbar puncture in the presence of papilledema is dangerous

Roentgenography —The specialist continues the examination by taking *radiographs of the skull* These may reveal a shift in the pineal shadow increased local vascularity and enlargement of the diploic veins erosion of the sella changes in the pituitary fossa destruction of the clinoids and erosion of the floor *Ventriculography* and *encephalography* aid in localiza tion The former involves withdrawal of fluid from the ventricles and the introduction of helium or oxygen for contrast radiography The latter is accomplished by doing a lumbar puncture and injecting air into the sub arachnoid space with similar intent There seems a general preference for ventriculography rather than encephalography although both procedures are accomplished at a definite risk

Electro encephalography —Electro-encephalography promises more ac curate localization of cortical and subcortical growths Characteristic changes found in the waves produced by the electrical current have diag nostic and localization value

Exploratory Craniotomy —There are times when even the expert can

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CON ED

tion of the spinal fluid As a result of these multiple possibilities the syndrome of spinal cord tumor is varied and unpredictable The clinical manifestations may be sensory or motor and in either instance may be irritative or paralytic

Pain Rigidity and Percussion Tenderness—Pain and paresthesias are very constant early manifestations of cord tumor particularly when there is involvement of the *posterior roots* Most often the pain is severe and of neuralgic distribution along the involved peripheral nerve The distribution may be unilateral or girdle like At times it simulates *visceral pain* so that exploratory laparotomy is performed for a suspected intraperito-

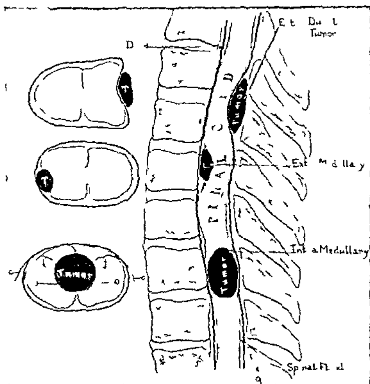


Fig 274—Schematic drawing to show the relation of tumors to the spinal cord and dura

neal disorder (p 1748) Pains in the *lumbar region* (p 2274) are difficult to differentiate from *low back sprains* and *sciaticas* particularly those associated with *herniation of the nucleus pulposus* (p 3074)

The pain of cord tumor is often exaggerated by sneeze cough or straining at stool The overlying spinal muscles are rigid as in arthritis and there may be *localized percussion tenderness* suggesting an osteomyelitis of the vertebral column

Sensory Disturbances—Sooner or later the pain of spinal cord tumor is associated with irritative or anesthetic manifestations Quite characteristically there is often a *band of hyperesthesia* just above the level of the

Stookey in Christopher Textbook of Surgery

neither sustain nor refute the possible diagnosis of brain tumor Under these circumstances it is necessary to do an *exploratory craniotomy* since the operative procedure has little risk and procrastination may cost the opportunity for complete removal of the neoplasm

Brain Abscess—The differential diagnosis of brain abscess (p 1469) is an academic discipline to the practitioner since the inflammatory tumor calls for surgical intervention as surely as a neoplasm

Vascular Disease—Cerebrovascular accidents and *aneurysms of the intracranial vessels* (p 1444) closely simulate expanding tumors especially those in which hemorrhage has occurred A reasonable doubt justifies *exploratory craniotomy* whose commission is a lesser risk to the patient with blood vessel disease than is omission for the person afflicted with the neoplastic disease Cerebral arteriography is of definite value in the recognition of the aneurysm

Treatment—The treatment of brain tumors is surgical As soon as the diagnosis is fairly certain or even suspected an *exploratory craniotomy* is advised by a *specialist neurosurgeon* The tumors that respond most favorably are acoustic neurinomas accessible meningiomas certain pituitary adenomas neurofibromas psammomas and certain gliomatous tumors of the cerebellar hemispheres

Roentgen therapy is indicated for spongioblastoma medulloblastoma glioblastoma astrocytoma pituitary adenomas and all infiltrating tumors

NEOPLASMS OF THE SPINAL CORD

Neoplasms of the spinal cord may be intramedullary or extramedullary The latter occur intradurally and extradurally The most frequent of the spinal cord tumors is the *primary extramedullary meningeal fibroblastoma* This neoplasm arises from the meninges and the sheaths of the nerve roots The more frequent *primary intramedullary tumors* are the *meningiomas* and *perineural fibroblastomas* From the viewpoint of operability it is important to stress the fact that 85 per cent of cord tumors are benign and amenable to excision

Secondary implants are usually the result of metastatic carcinoma involving the female breast the stomach the prostate gland or the large intestines

Clinical Manifestations—Cord tumors occur most commonly between the ages of twenty and fifty but they may develop in young children and the aged The size of the tumor varies from that of a pea to that of a large nut Giant tumors may extend up and down the vertebral canal for a considerable distance hour glass neoplasms grow partly inside and partly outside the spinal canal the waist line being in the intervertebral foramen

The effect of the neoplastic growth is variable and depends upon chance factors Compression of the posterior roots produces early *sensory* manifestations pressure anteriorly involves *motor* areas the cord may be pushed to one side or compressed, the symptoms may result from contrecoup the vascular supply may be cut off leading to secondary cord softening and degeneration the veins may become engorged and varicose the spinal fluid may be blocked with the production of the *Froin Nonne syndrome* of *xanthochromia, increased protein* and *spontaneous coagula*

tion of the spinal fluid As a result of these multiple possibilities the syndrome of spinal cord tumor is varied and unpredictable The clinical manifestations may be sensory or motor and in either instance may be irritative or paralytic

Pain Rigidity and Percussion Tenderness—Pain and paresthesias are very constant early manifestations of cord tumor particularly when there is involvement of the *posterior roots* Most often the pain is severe and of neuralgic distribution along the involved peripheral nerve The distribution may be unilateral or girdle like At times it simulates *visceral pain* so that exploratory laparotomy is performed for a suspected intraperito

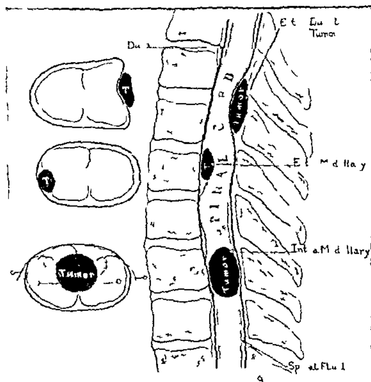


Fig 274—Schematic drawing to show the relation of tumors to the spinal cord and dura

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Hematopoietic	Particularly with hyperchromic macrocytic anemia (pernicious anemia) It is characteristic hemogram (p 1077) and favorable response to liver extract (p 1078)
Pachymeningitis and Arachnitis	Usually secondary to organization of meningeal clots following hemorrhage Simulation of cord tumor discernible only at laminectomy (p 1448)
Myelopathies	Rare metabolic lesion secondary to vitamin deficiency hyperchromic macrocytic anemia or poisoning with carbon monoxide carbon sulfide or chloroform
The Scleroses	Idiopathic lesions of posterior or anterior horns posterior columns or pyramidal tract Diffuse involvement precludes possibility of operative interference Temporal pallor of optic disks in multiple sclerosis (p 1502)
Syringomyelia	Central gliosis with sensory dissociation Contralateral loss of pain and temperature with ipsilateral touch anesthesia (p 1505) Diffuse changes preclude surgical interference

lesion Below this there may be *segmental hypesthesia or anesthesia* A *saddle anesthesia* suggests a lesion of the conus

Involvement of the *spinothalamic tract* gives rise to a *contralateral analgesia* and *thermanalgesia* with resultant burns and *trophic ulcers* produced by heat that had been locally applied for the relief of pain

Motor Disturbances—Pressure of a cord neoplasm on an *anterior root* results in motor weakness followed by atrophy and loss of reflex Should the *pyramidal tract* be involved the limb is spastic deep reflexes are increased the Babinski and confirmatory signs are positive and cremasteric and abdominal reflexes are lost Whether the motor paralysis is flaccid or spastic study of the innervation of the affected muscles assists in determining the tumor level

Visceral Disturbances—Difficulties in urination defecation ejaculation and erection are dependent upon the degree of cord compression and the level of the tumor The initial disturbance is usually a disturbance in *initiating urination* Later there may be *retention* and *overflow incontinence* (p 2265) Difficulties with *defecation* are often early manifestations of compression of the conus Irritative phenomena in the lower cord produce *priapism* but more extensive involvement is followed by *impotence* and *sterility*

Determination of Tumor Level—The practitioner is not required to define with exactitude the level of a cord neoplasm He has fulfilled his purpose if he recognizes the presence of the growth and refers his patient to the neurosurgeon The latter assumes the responsibility of determining the site for his laminectomy and even the expert may be considerably misled by anomalies and contrecoup phenomena

Tumors of the Conus—Tumors of the conus which carry a bad prognosis give early bilateral cord symptoms saddle anesthesia trophic disturbances and marked involvement of the bladder and rectum They are apt to produce symmetrical manifestations with *little pain*

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Spinal Cord

Disturbances of the spinal cord are complicated by the fact that they may arise secondary to derangements involving the meninges and the osseous and articular structures that make up the vertebral column

CAUSE	DIAGNOSTIC FEATURES
Congenital and Hereditary Abnormalities	Spina bifida Note manifest defect or obtain x ray for occult anomaly (p 1411) Hereditary spinal and cerebellar ataxia (p 1415)
Neoplasms	With pain rigidity and localized percussion tenderness Localizing sensory and motor disturbances Refer to neurosurgeon for x ray laminography spinal manometry and examinations of fluid particularly for xanthochromia (p 3735)
Vertebral Osteopathy	With metastatic carcinomatosis of spine (p 2836) With osteomyelitis usually tuberculous or pyogenic (p 2930) Note changes in x ray
Osteoarthrosis	Metabolic disturbance of obese middle-aged Note narrowing of joint spaces and spur formations producing irritations of spinal roots particularly of sciatic distribution (p 2855)
Atrophic Arthritis of the Spine	Generalized arthropathy of young with narrowing of joint spaces and tendency to ankylosis (p 2859) Note rapid sedimentation rate and peripheral vasomotor disturbances
Trauma	Lesions secondary to fracture dislocation of spine or transection of cord (transverse myelitis) History of injury X ray changes and flaccid or spastic paralyses (p 14[illegible]6) Often associated with disturbances of bladder and rectum
Herniation of Nucleus Pulposus	History of acute chronic or recurrent radiculitis usually of sciatic distribution Defect demonstrable by laminography and laminectomy (p 3704)
Tabes Dorsalis	Syphilitic meningomyelitis with pupillary changes disturbances of deep sensation and characteristic spinal fluid findings Positive serology and decolorization of intermediate tubes in colloidal gold reaction (p 1465)
Acute Anterior Poliomyelitis	Acute virus infection with fever spinal pleocytosis and peripheral nerve palsies usually flaccid (p 4[illegible]7)
Nonsuppurative Meningitides	Most often tuberculous with acid fast bacilli demonstrable in spinal fluid (p 1462) May be of virus origin in lymphocytic chorio-meningitis and equine encephalomyelitis (p 442)
Suppurative Meningitides	With demonstrable organisms in meningococcal streptococcal, staphylococcal pneumococcal and influenzal invasions (p 1462)

CONTINUED

NEOPLASMS OF THE PERIPHERAL NERVES

The peripheral nerves may be involved in neoplastic processes References are made elsewhere to *acoustic neurinomas* (p 1426) and similar lesions which are often part of a *generalized neurofibromatosis* (von Recklinghausen s disease) (p 1415)

Glomus Tumors—Neoplasms may involve the neuromyo arterial glomus The growths appear as *bluish or purplish nodules* that are most often situated *subungually* Attention is drawn to the lesion by severe and intractable *pain* and exquisite *tenderness* Glomus tumors may appear anywhere in the corium of other parts of the hands and feet

Simple *excision* is followed by immediate relief of symptoms

Tumors of the Cauda Equina—Tumors of the cauda equina which have a more favorable outlook than those that involve the conus are apt to be characterized by *severe pain* retention of bladder and rectal control early loss of knee and ankle jerks and a symmetrical distribution of motor and sensory manifestations

Laboratory Findings—The problem of diagnosis of spinal cord tumor is greatly clarified by radiography manometry and examinations of the spinal fluid

Roentgenography—Upon suspicion of the presence of a cord tumor *a radiograph of the vertebral column is taken for the possible demonstration of a local lesion which may be responsible for the cord disturbance* The frequently encountered abnormalities include fractures and dislocations caries of the spine usually tubercular an osteo arthrosis with spur formation and osteophytes the atrophic type of spondylitis an osteomyelitis a primary or metastatic malignancy or a spina bifida occulta

Queckenstedt s Test—After the radiographs of the vertebral column have been taken a lumbar puncture is performed and *spinal fluid pressure readings* are made with and without compression of the veins in the neck In the recumbent position the normal pressure of the cerebrospinal fluid is 10 to 15 mm of water *Compression of the jugular vein on both sides* of the neck normally is followed by a rise in pressure up to 35 to 50 mm of water Release of the vessels is accompanied by a sharp fall to the previous level When there is obstruction of the subarachnoid space the pressure reactions are absent or delayed

Examination of the Spinal Fluid—At the termination of the manometric readings a *xanthochromic fluid* with an increase of globulin and *spontaneous coagulation* points to a tumor *above the level of the tap* with a fairly complete block of the spinal subarachnoid space The usual examinations are then performed as to cytology albumin and sugar content serology bacteriology and the colloidal gold phenomenon In the presence of a significant lymphocytosis leukocytosis positive serological test for syphilis changes in the colloidal gold reading or a positive growth of a bacterium attention is directed toward conditions other than the spinal cord neoplasm Contrariwise *if all of these tests are negative* but the globulin content is elevated and definitive organic neurological findings are demonstrable the suspicion of cord tumor cannot lightly be dismissed

Treatment—The treatment of the cord tumor is approached through a *laminectomy* and operative removal of the neoplasm if it is at all feasible The expert neurosurgeon is often rewarded by the accomplishment of a complete excision of a meningioma a fibroma a neuroma a neurofibroma and of certain types of sarcoma

Inoperable tumors are treated by a *decompressive laminectomy* *Roentgen therapy* may be of some palliative value in the management of various types of sarcomas intramedullary astrocytomas ependymomas and metastatic lesions Symptomatic surgical therapy for the relief of pain consists of *chordotomy* retention of urine is best managed by a *suprapubic cystostomy* preceded by ligation of the vasa deferentia

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CHAPTER 71

THE VOLUNTARY NERVOUS SYSTEM CIRCULATORY DISTURBANCES

CIRCULATORY disturbances of nervous tissue have the capacity for the production of severe and often irreparable damage Nerve tissue suffers profound change as the result of *local ischemia* from whatever cause *regeneration* is imperfect if it occurs at *all Circulatory disturbances* characterized by hemorrhage and clot formation encroach upon the limited confines of intracranial and intravertebral spaces and result in *compression manifestations* that are identical with those produced by *neoplasms* (p 1419) Traumatic disturbances are elsewhere described (p 1450)

CEREBRAL ANEMIA

Cerebral anemia may be due to systemic or local conditions The *acute anemia of hemorrhage* (p 1057) is characterized by noises in the ears spots before the eyes yawning sighing and syncope Similar symptoms occur in *shock* (p 928) when there is no actual blood loss

The *chronic anemias* (p 1055) are often associated with headache dizziness roaring in the ears loss of memory insomnia drowsiness delusions hallucinations and headache Extraordinary recovery is the rule when blood volume is restored

Local Cerebral Anemia and Intermittent Claudication—A local cerebral anemia may be due to organic closure of an arterial lumen as described in the paragraphs on thrombosis (p 1123) A similar reaction pattern may be caused by an angiospasm as described in the following paragraphs

Intermittent Claudication—Intermittent claudication of the cerebral vessels may result in transitory aphasia paresis paralysis convulsive twitchings hemianopsia or sensory disturbances These may be *angioneurotic* (p 3349) as well as harbingers of *cerebral arteriosclerosis* (p 1438) *Sensitization to tobacco* (p 3884) may be a factor in isolated instances In the interests of therapy and diagnosis an intravenous injection of

papaverine hydrochloride 32 to 65 mg (½ to 1 grain) is indicated with repetition in 1 to 2 hours

Diagnosis and Treatment —The diagnosis of cerebral angiospasm should not be hazarded until efforts are made to exclude the pre ence of organic

DIFFERENTIAL DIAGNOSIS OF

Non Traumatic Abnormalities of Cerebral Circulation

See also Traumatic Lesions (p 1450)

	Etiology	*Age*	*Comment*
Cerebral Anemia	Blood loss Forward Failure	Any	Relief by correction of etiologic cause
Cerebral Angio-*spasm*	Allergy Tobacco	Young	Intermittent spasms with recur rences on repeated exposure to allergen (p 547)
Cerebral Hyperemia	Backward failure Pol ycythemia vera Hy pertension. Glut tony Sunstroke	Any	Relief by correction of etiologic agent
Cerebral Edema	Head injury Alco holism Metabolic disturbances such as uremia	Any	Increased intracranial tension re lieved by lumbar puncture and intravenous injection of hyper tonic sugar
Cerebral Arterio-sclerosis	Diffuse vascular dis ea e	After 50	Progressive deterioration
Cerebral Hemor rhage	Cerebral arterioscle rosis	After 50	Acute stroke or shock, usually with hemiplegia
Cerebral Throm bosis	Cerebral arterioscle rosis	After 50	Disability may be ingravescent with slowly progressing manifestations usually hemiplegia
Cerebral Emboliza tion	Pleuropulmonary in fection Coronary thrombosis	Any	Sudden onset in patient previously incapacitated by primary dis ord r
C rebral Aneurysm	Congenital malforma tions	20 to 40	May be treated surgically
Subarachnoid Hem orrhage	Arteriosclerosis an eurysm or syphilis	Any	Cerebrospinal fluid bloody or xan thochromic (p 3735)
Thrombosis of the Venous Sinuses	Contiguous infection	Young	May be associated with kull frac ture face infection or inflamma tions of eye ear or nose

disease If the afflicted person is young free from arteriosclerotic change nonsyphilitic with normal intracranial pressure and an otherwise normal neurological status continued conservative observation is warranted An investigation is made for *allergic sensitivities* (p 558) *smoking* is for bidden entirely and forever

CEREBRAL HYPEREMIA

Chronic passive congestion of the cerebral vessels occurs in *backward circulatory failure* (p 941) particularly when associated with *hypertension* and/or *polycythemia* (p 1092) Patients complain of feelings of warmth flushing of the face throbbing in the temples dizziness and headache there may be insomnia injection of the sclerae slowing of the pulse and respiratory irregularities

Active congestion is associated with excitement gluttony bibulousness or with sunstroke It produces pounding in the head dizziness and transitory lapses of memory or consciousness Hyperemic symptoms occur in *acute polycythemia* whether physiological as at high altitudes or pathological as in Osler Vaquez disease (p 1092)

Treatment—Cerebral hyperemia is ordinarily relieved by simple measures such as avoidance of the provocative factors of hyperalimentation and alcoholism A forced *emesis* may be salutary and a *saline purge* produces rapid dehydration The application of *cold compresses* or an *ice cap* to the skull while the patient is recumbent is often highly effective for symptomatic relief Those who develop symptoms at a high altitude are instructed to take inhalations of *oxygen* until they can return to points nearer sea level

The old remedy of a liberal *blood letting* is still highly to be recommended and may serve prophylactic and psychologic purpose in plethoric high livers

CEREBRAL EDEMA

Cerebral edema or *wet brain* is often associated with *head injury* (p 1450) *alcoholism* (p 3851) and *endogenous intoxications* such as *a otemia* (p 2275)

Intracranial pressure may be reduced with rapidity by repeated injections of 50 to 200 cc of 50 per cent *dextrose* or *sucrose* by *lumbar puncture saline catharsis* and a *dehydration diet* (p 664)

CEREBRAL ARTERIOSCLEROSIS

Cerebral arteriosclerosis may be a relatively limited process or a local manifestation of a generalized hardening of the arteries The resultant disturbances are varied and range from minor manifestations to major afflictions such as *cerebral apoplexy* (p 1439) *cerebral thrombosis* (p 1444), the *presenile psychoses* (Pick Alzheimer) or the *senile* and *post apoplectic psychoses* (p 1382)

Autopsies usually reveal much more extensive change than had been suspected by the clinical manifestations the vessels are thick walled and contain arteriosclerotic plaques the convolutions are atrophied the meninges are thickened and the glial substance is increased

Clinical Manifestations—A cerebral arteriosclerosis may be the fundamental lesion responsible for mild dizziness transitory attacks of syncope occasional episodes of nausea and vomiting fleeting paralyses aphasia or dysphonia There may be an *intellectual deterioration* progressing to obvious *dementia* Occasionally the syndrome of *pseudobulbar palsy* is noted with dysarthria difficulty in mastication dysphagia a small step-

page gait emotional instability and drooling *Characterological changes* include irritability excessive or flattened emotions suspicion slovenliness sexual exhibitionalism and senile leering

Rupture of a sclerotic vessel produces the manifestations of an *apoplexy* (p 1439) *thrombotic occlusion* gives a similar clinical picture (p 1444) an *aneurysmal dilatation* may suggest a brain tumor (p 1419) and free bleeding results in *intraventricular* or *subarachnoid hemorrhage* (p 1441) The *senile dementias* are described with the material on psychiatry (p 1382)

Treatment —See *Arteriosclerosis* (p 979)

CEREBRAL HEMORRHAGE (APOPLEXY)

Cerebral hemorrhage or apoplexy results from rupture of a vessel whose wall has undergone local or diffuse pathologic change The damage to the surrounding brain tissue does not differ significantly from that caused by the more frequent *cerebral thrombosis* (p 1444) *cerebral embolization* (p 1444) or bleeding into a *neoplasm* (p 1430) The present discussion may be regarded as applicable to all types of intracerebral vascular accident later material deals with the differences displayed in thrombotic and embolic disturbances

Etiology —The commonest cause for cerebral apoplexy i *arteriosclerosis* of a vessel wall (p 1438) The degenerative process is almost always patchy and may involve the cerebral vessels to greater or lesser degree than those of the general circulation More particularly it may affect a single cerebral artery in a manner disproportionate to the involvement of other intracranial arteries Despite the general contrary opinion cerebral apoplexy is *rarely syphilitic* and most often is associated with *cardiovascular renal disease* and/or *hypertension*

The immediate *precipitants* of an apoplexy may be sudden increases in intracranial tension due to straining at stool excitement coughing vomiting parturition or coitus The syndrome is more frequent in those of the *apoplectic habitus* observed in plethoric individuals who are thickset and short necked (p 3492)

Clinical Manifestations —The apoplectic stroke is often preceded by *premonitory symptoms* such as dizziness headache a sensation of fulness in the head spots before the eyes and anxiety There may be the transitory localizing symptoms of dysarthria muscle weakness aphasia paresthesias vomiting or retinal or sub conjunctival hemorrhages

The Stroke or Shock —The actual stroke or shock sets in suddenly with rapidly developing *coma* and *loss of consciousness* During this period the patient lies limp the face is flushed breathing is stertorous and irregular the pulse rate is slowed the pupils may be dilated but usually react to light corneal sensation is lost deep and superficial reflexes are abolished there may be retention or incontinence of urine and feces the voided urine often contains albumin and sugar and the temperature reading may show an elevation or a hypothermia

Localizing Signs —The *duration* of coma is variable and depends upon the extent of the hemorrhage Sooner or later there appear localizing manifestations such as ballooning of the cheek of the paralyzed side pupil

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peduncles produces the syndrome of Weber with ipsilateral third nerve paralysis and contralateral hemiplegia *Pontine bleedings* are followed by ipsilateral cranial nerve paralysis contralateral limb involvement and crossed anesthesia in which the ipsilateral face and the contralateral body are affected *Bulbar palsies* occur with medullary lesions

Intraventricular Hemorrhage—Intraventricular hemorrhage is characterized by deepening coma pupillary miosis increasing paralysis and rigidity of the involved extremity stiffness of the neck convulsive movements in the limbs generalized decerebrate convulsions a bloody spinal tap marked slowing of pulse and respiratory rates pre agonal hyperthermia and a rapidly fatal termination

Differential Diagnosis—The differential diagnosis of a cerebral hemorrhage during the phase of unconsciousness is discussed with the broader considerations of the management of *coma* (p 1295)

Hemorrhage or Thrombosis—Until the present availability of *anticoagulants* (p 1045) in the therapy of vascular occlusions it was an academic discipline to attempt to differentiate *cerebral hemorrhage* from *cerebral thrombosis* (p 1444) Since the latter condition may be phenomenally alleviated by *heparinization* (p 1050) whereas a bleeding tendency might be seriously augmented by the intended therapeutic measure exact diagnosis now has practical therapeutic importance It is unfortunate that there is no available method of clearly distinguishing cerebral thrombosis from apoplexy However in favor of hemorrhage and opposed to thrombosis are the undernoted clinical phenomena

1 The presence of blood or xanthochromia in the cerebrospinal fluid
2 More sudden onset with headache vomiting or convulsions
3 Rigidity of the neck and the presence of the Kernig sign
4 Unilateral dilatation of a pupil
5 Conjugate deviation of head and eyes
6 A bilateral Babinski reflex
7 Progression of the focal neurological signs
8 A leukocytosis in excess of 12 000 per cubic millimeter
9 A cerebrospinal fluid pressure reading in excess of 350 or 400 mm

Brain Tumor or Abscess—In the stage of paralysis it becomes necessary to consider the possibility that the focal neurologic manifestation originates in a lesion that is operable such as a *brain abscess or neoplasm* (pp 1419 1469) There also remains the eventuality of a coexistence of hemorrhage and neoplasm since bleeding may occur within a new growth The existence of a brain tumor or abscess is favored when the patient is relatively young and there are few manifestations of arteriosclerotic disease elsewhere in the body Abatement of the paralytic phenomena argues strongly towards the diagnosis of bleeding persistence or increase in focal signs warrants consultation with the expert neurologist or neurosurgeon who may require *exploratory craniotomy* before a definitive opinion can be established

Prognosis—The prognosis of a cerebral apoplexy is grave but by no means hopeless The majority of the patients survive the accident though few are enabled to resume the normal activities of an average life During

lary contraction in pontine and intraventricular bleedings contralateral abolition of the corneal reflex in supratentorial hemorrhages jacksonian epilepsies conjugate deviations of the head and eyes to the side of the bleeding and away from the paralyzed side obvious weakness of an arm and/or leg or ipsilateral papilledema The superficial abdominal and cremasteric reflexes fail to return on the paralyzed side a Babinski sign is elicited even in the early stages of coma

Stage of Recovery—With minor apoplexies the patient recovers after the lapse of minutes hours or days A fortunate few have no localizin

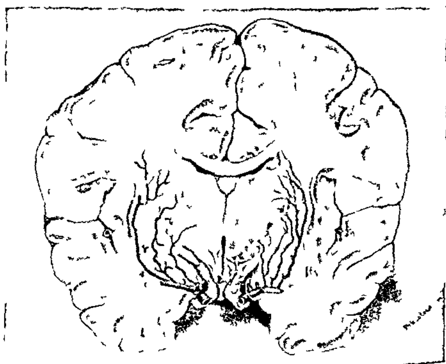


Fig 27b —Diagram showing the branches of the middle cerebral arteries which supply the basal ganglia The innermost are the lenticulo-optic the outer two on each side of the lenticulo striate arteries The outermost of these which pierces the internal capsule to end in the caudate nucleus is known as the artery of cerebral hemorrhage

manifestations but the majority of the afflicted progress to the phase of paralysis

The Stage of Paralysis—The paralytic phenomena that follow cerebral apoplexy depend upon the localization of the hemorrhagic accident Since the involved vessels are most often the lenticulostriate and the lenticulo optic arteries with predominant bleeding into the internal and external capsules there is most often a resultant *hemiplegia* The involved limbs are *flaccid* for several weeks before the *spastic phenomena* of upper cortical neurone involvement become apparent Associated *sensory disturbances* point to a lesion in post central cortical or subcortical areas posterior portion of capsule or thalamus *Hemorrhage into the cerebral*

* MacCallum mod fed from Charcot Duret

nothing to be gained by administering brandy or whiskey or by injections of caffeine camphor epinephrine digitalis strychnine coramine or alpha lobeline The use of sedatives such as chloral bromides opiates and barbiturates is equally ill advised and may cloud significant diagnostic information *Since on rare occasions the hemorrhage may be idiopathic or* a herald manifestation of a tumor consultation with the neurosurgeon merits consideration

Management of the Phase of Paralysis—During the first few days following the accident phlebotomies and intravenous injections of sucrose are repeated according to indication Fluids are administered by rectum or through an intravenous drip urinary secretion is drained off by catheter when necessary No attempt is made to maintain nutrition or disturb the patient by attempts to evacuate the bowels Hypostasis is prevented by turning the patient frequently from side to side The skin is protected by expert nursing which aims at the maintenance of dryness the prevention of soiling and easing of all pressure points

When coma lightens and the patient reacts teaspoonful doses of water are administered Larger amounts are given if swallowing can be accomplished without gagging and coughing The paralyzed extremities are splinted to prevent spastic deformities and contractures

When the patient has thoroughly recovered from the initial shock it is wise to put the joints through their ranges of passive motion These measures have psychotherapeutic value for the patient who is eager to speed convalescence and frets that nothing is being done

As soon as voluntary motion returns the patient is encouraged to focus on active therapy a rubber ball is placed in the hand to aid in the return of strength and dexterity toes and fingers are put through their arcs of motion several times daily *We are of the opinion that electrotherapy* has no beneficial effect and may be harmful in the presence of spasticity

As soon as possible the patient is permitted to sit at the bedside and use a bedside commode Persistent encouragement on the part of the physician is required and *daily visits* are almost a necessity If the physician can think of no other excuse for his continued presence he is justified in giving injections of sodium cacodylate neurophosphates or vitamins in order that *supportive psychotherapy* (p 1316) be sustained Failure to make frequent and repeated visits leads the patient to assume that the condition is hopeless and adds the element of despair to the circulatory accident

During his visits the physician functions as a *physiotherapist* He conducts resistance exercises in the normal ranges of joint motion and later accompanies his patient on trips to the bathroom the living room down the steps out of the house and to the street If the prophylactic routine (p 1442) has not been instituted before the cerebral accident it is started immediately after recovery in the hope of preventing recurrence

As a rule the apoplectic begins to walk within six to eight weeks and recovery may continue for the period of another year Intelligent and skilful reeducation with a modification of the *way of life* (p 3473) accomplishes much that is good A philosophical patient adjustment may permit a peaceful and comfortable survival for a long period of time

the phase of coma *ominous prognostic signs* include persistence of un*consciousness* beyond the first twenty four hours evidences of bleeding into ventricular cavities pons or medulla hypopyrexia or hyperpyrexia bradycardia or tachycardia depression or increase of respiratory rate the onset of Cheyne Stokes respirations a prolonged period of flaccidity of the affected area continued absence of reflexes or an excessively rapid onset of spasticity

Treatment—Prophylactic and symptomatic measures are available in the management of a cerebral apoplexy

Prophylaxis—The prevention of cerebral apoplexy involves the institution of measures employed in the management of *arteriosclerosis* (p 979) *Weight reduction* is mandatory in the obese (p 697) *phlebotomy* is advisable in the plethoric and those who have a polycythemia (p 1092) *moderation* is required in eating and in physical and emotional activities *drinking* and *smoking* are best given up entirely *sedatives* (p 3837) and *hypnotics* (p 3837) are prescribed for tense highstrung individuals the judicious use of *thyroid extract* (p 1189) to aid in weight reduction is suggested in those who have a low basal metabolic rate whether or not there are signs of myxedema social activities are curtailed the amount of work is reduced to that which is absolutely essential for sustenance and the maintenance of self respect

The administration of *iodide* (p 608) accomplishes nothing in our opinion, beyond untoward symptoms *antisyphilitic therapy* (p 340) is reserved for those who are demonstrably luetic and should not be administered on mere pragmatic grounds

The Management of Coma—The immediate treatment of the patient with an apoplexy involves the application of the principle of *skilful neglect* If it is at all possible the patient is not moved or disturbed except for loosening clothes If transportation is required expert handling does much to prevent more excessive damage to delicate nerve tissue

Plethoric individuals benefit by a generous *venesection* (p 3780) intracranial pressure is reduced by *intravenous injections* of 50 to 200 cc of 50 per cent *sucrose* (p 3773) *lumbar puncture* is not without danger but is required for diagnostic purposes under which circumstance a minimal quantity of fluid is withdrawn

The application of an *ice cap* to the head can do no harm but probably has little effect other than to give the agitated bystander something to do The bladder region is carefully watched overdistention is prevented by frequent *catheterization* or the temporary use of an *indwelling catheter* Body heat is maintained by blankets but great caution is exercised to prevent burns from awkwardly applied electric pads or hot water bottles Prophylactic therapy with penicillin (p 106) is advised

If there is a suspicion of an angiospastic element an intravenous injection of *papaverine hydrochloride* 30 to 60 mg (½ to 1 grain) is worthy of trial The drug may be repeated within two hours but efforts are best abandoned if two injections fail to cause demonstrable beneficial effects Despite enthusiastic reports we have had no success with the intravenous use of *aminophylline* 0 3 to 0.5 gm (5 to 7½ grains) which has the advantage that it can cause no harm

The apoplectic is often traumatized by overzealous therapy *There is*

branch to the ipsilateral eye and lies in intimate relationship with the cavernous sinus the third fourth sixth and the ophthalmic divisions of the fifth cranial nerve the sella turcica and the optic chiasm

Clinical Manifestations —The clinical manifestations of the cerebral aneurysm rarely appear before the age period of fifteen to twenty Most commonly they are noted in the third and fourth decades when the aneurysm undergoes dilatation and produces symptoms simulating those of an intracranial neoplasm (p 1419) At this time there may be *bitemporal headache* followed by pressure symptoms resulting from involvement of the surrounding structures Partial or complete *external ophthalmoplegia* suggests pressure on the sixth nerve a *facial neuralgia* or *anesthesia* is noted with involvement of the first branch of the fifth nerve When the aneurysm is located along the course of the posterior communicating artery an *oculomotor paralysis* becomes manifest enlargement of the sylvian artery as it enters the parenchyma of the brain sets up foci of cortical irritation and *epileptogenic manifestations* erosions into the sphenoid sinus with thrombosis of the cavernous sinus produce an ipsilateral *pulsating exophthalmos* and *papilledema* radiographs may show *erosions of the ipsilateral posterior sphenoid process* and a curvilinear streak just above and lateral to the sella due to calcification within the arterial wall

Sooner or later there is thrombus formation with gradual reduction of the vascular lumen until the carotid is entirely occluded A *slow blockage* may not be associated with clinical manifestations since there is opportunity for the opposite carotid to undergo compensatory enlargement and supply both sides of the circle An *acute occlusion* produces the syndrome of a *cerebral apoplexy* or *thrombosis* (p 1439) Rupture of the aneurysm produces the signs and symptoms of a *subarachnoid hemorrhage*

Treatment —The treatment of intracranial aneurysms involves a *surgical approach* to the internal carotid artery The application of slow compression and eventual ligation of the extracranial portion are the only possible preventive measures for this otherwise fatal condition

SUBARACHNOID HEMORRHAGE

Bleeding into the subarachnoid space results from trauma or rupture of a damaged cerebral vessel weakened as the result of arteriosclerosis syphilitic endarteritis or aneurysmal dilatation

Pathology —In subarachnoid hemorrhage the blood mixes with the cerebrospinal fluid and produces *irritations* of the *cerebral cortex* and the *posterior roots* The extravasated blood clot later serves as a space filling tumor producing the syndromes of *intracranial neoplasms* (p 1419) With complete healing multiple adhesions set up a *pachymeningitis* (p 1448) or an *adhesive arachnitis* (p 1448)

Clinical Manifestations —An intracranial subarachnoid hemorrhage is announced by *vomiting* severe *head pain* followed by *coma* and *collapse* in the manner of a cerebral apoplexy (p 1439) Irritation of the cortex when the blood breaks through the pia may produce *convulsive phenomena* or the patient may display the manifestations of *increased intracranial pressure* (p 1491)

CEREBRAL THROMBOSIS

Cerebral thrombosis is a more common cause than an apoplexy for the acute intracerebral vascular accident Most often the arterial occlusion is on the basis of an *arteriosclerotic intimal involvement* but it may also occur in the course of an acute illness such as typhoid fever diphtheria or pneumonia it may complicate a surgical procedure or a chronic affliction such as polycythemia vera or the cachexias of malignant disease

Clinical Manifestations—The clinical manifestations of cerebral thrombosis are those of the apoplexy The most striking differential feature is noted in the *ingravescent type* of disturbance in which symptoms are manifest for several days preceding actual loss of consciousness or the onset of paralytic phenomena As in the instance of the cerebral hemorrhage the favored locations are the lenticulostriate or lenticulo optic vessels The clinical phenomena are identical with the exceptions noted in the discussion of the differential diagnosis of cerebral hemorrhage and cerebral thrombosis (p 1441)

Treatment—The course and prognosis of cerebral thrombosis do not differ from those of cerebral apoplexy

Anticoagulants—If the physician can be assured that there is no bleeding and suspects that the vascular occlusion is on a thrombotic basis the use of *anticoagulants* using oral dicoumarol or injections of *heparin* (p 1050) may be attempted for dissolution of the clot and prevention of the spread of the process Because of the dangers involved in heparinization (p 1051) and the present experimental status of therapy this procedure is not advised unless the patient is *institutionalized* and under complete and careful observation It is *contraindicated* if there are obvious evidences of bleeding such as hemorrhagic spinal fluid or xanthochromia (p 3735)

CEREBRAL EMBOLIZATION

Cerebral embolization is a variety of vascular accident that is more apt to occur in young people who suffer from pulmonary and pleural infections and the vegetative types of *endocarditis* (p 1021) In older patients it is observed most commonly following a *coronary thrombosis* (p 983)

Anticoagulants—The presence of the cerebral embolus is suspected when the vascular accident occurs as a complication of the clinical conditions previously listed The recognition of the mechanism of the vascular injury assumes therapeutic importance since *heparinization* (p 1050) may be instituted with the hope of minimizing the damage to brain tissue just as in the instance of cerebral thrombosis (p 1444)

Septic Emboli—Septic emboli may produce abscessed areas in the brain (p 1469) If their presence is suspected *heparinization* is accompanied by the administration of *sulfonamide* or *penicillin* (p 106) for prophylaxis and active treatment

CEREBRAL ANEURYSMS

Cerebral aneurysms occur as *congenital structural malformations* The defect in the vessel wall usually is encountered along the intracranial course of the carotid arteries at their junctions with the anterior portion of the circle of Willis This area of the carotid gives off an ophthalmic

branch to the ipsilateral eye and lies in intimate relationship with the cavernous sinus the third fourth sixth and the ophthalmic divisions of the fifth cranial nerve the sella turcica and the optic chiasm

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The diagnosis is established with certainty if the *spinal fluid* reveals evidence of gross bleeding With the cessation of active hemorrhage the fluid becomes *xanthochromic* the protein content is increased and a *pleo cytosis* is observed

Course and Treatment —The course of a subarachnoid hemorrhage is not unlike that of any other type of cerebral accident There may be complete recovery a fatal termination or survival with residual paralyses

Lumbar Puncture —The advisability of performing *lumbar puncture* during the period of active bleeding is seriously debated The advocates are impressed by the reduction of the increased intracranial tension while the opponents fear perpetuation of the bleeding We favor a diagnostic tap *The decision regarding repetition depends on the patient s reaction to* the first puncture An improvement in symptoms justifies continuation whereas increased difficulties serve as a contraindication Anticoagulants are contraindicated Consultation with the neurosurgeon is imperative

THROMBOSIS OF THE INTRACRANIAL VENOUS SINUSES

The intracranial venous sinuses lie within the dura and receive blood from *intracerebral* and *extracerebral* vessels The most important are the superior longitudinal cavernous and lateral sinuses

Superior Longitudinal Sinus —The superior longitudinal sinus in its most anterior portion connects with the veins of the *nasal cavity* through the foramen cecum In its course between the superior portions of the cerebral hemispheres it receives venous branches from the *skull* and *scalp* *and drains venous blood from the neighboring mesial* and superior portions of frontal parietal and occipital lobes Posteriorly the superior sinus ends in the lateral sinus usually on the right side

Cavernous Sinus —The cavernous sinus is located about the lateral and posterior portions of the *sella turcica* It is formed by the *ophthalmic vessels* and the *angular veins* which drain the nose upper lip cheeks and upper teeth It has no important intracranial branches and unites with the *petrosal sinuses* which communicate with the lateral sinus and *the internal* jugular veins

Lateral Sinus —The lateral or *transverse sinus* is a paired structure which is located close to the inner ear and makes connections with the veins of the *mastoid bone* the *middle and internal ear* It also receives branches from the closely situated petrous bone and the lateral portions of the temporal lobe The paired lateral sinuses empty into the jugular veins by way of the sigmoid sinuses

Etiology —*Primary thrombosis* of a venous sinus is very rare and when present, is a component of the late stage of cachexia in the young or old

The most frequent cause of thrombosis of the venous sinuses is *infection* resulting from an inflammatory process in a nearby structure The commonest lesion is a *phlebitis* which arises in the extracranial areas and spreads to the communicating intracerebral veins and venous lakes The *secondary thromboses* cause venous occlusion and intracerebral disturbances such as edema perivascular inflammation encephalitis diffuse cortical and subcortical lymphocytic infiltration encephalomalacia multiple petechial hemorrhages or brain abscess The pathological changes may remain localized or involve an entire cerebral hemisphere

Clinical Manifestations —The clinical picture of intracranial sinus thrombosis consists of systemic symptoms and localizing manifestations that vary with the site of the involvement

Systemic Symptoms —The generic manifestations of septic endophlebitis include *chilling a hectic fever leukocytosis bacteremia petechial hemorrhages enlargement of the spleen* and *metastatic deposits in the lungs* Unless there is an associated meningitis the spinal fluid is clear and sterile a *lymphocytosis* suggests an impending subarachnoid involvement (*meningitis sympathica*) and positive cultures or a polymorphonucleosis point to an actual break in the meningeal barrier

Superior Longitudinal Sinus Thrombosis —Thrombosis of the superior longitudinal sinus is uncommon and its presence is usually masked by the signs and symptoms of the primary intracerebral lesion such as an abscess hemorrhage or neoplasm Less often it follows infections of the scalp or skull particularly in enfeebled infants

Locally there is *tenderness* over the vertex and an associated *edema* which may spread downward over the forehead The frontal and temporal veins are usually dilated the patient complains of *headache epistaxis nausea* and *vomiting* the associated encephalitis produces *lethargy drowsiness* or *convulsions* the *fontanelle* of the infant may bulge

Since the superior longitudinal sinus lies between the paracentral lobules of the brain which are the cortical centers for bladder control there is usually *incontinence of urine* Involvement of the motor and sensory cortex may produce *weakness or paralysis of one or both lower extremities* with or without impairment of the cortical sensory modalities in the lower extremities Other neurological signs and symptoms depend upon the location of the underlying intracerebral pathology The course of the affliction is rapidly downhill with death in a few days or weeks

Cavernous Sinus Thrombosis —The reaction pattern resulting from thrombosis of the cavernous sinus is fairly constant and usually follows an acute infection in the butterfly area of the face

The venous circulation of the orbit and face are impaired producing *periorbital edema chemosis proptosis congestion* and *dilatation* of the frontal and retinal veins and *papilledema* There may be relatively few associated symptoms but *headache diplopia nausea* and *vomiting* accompany the more severe infections Peripheral involvement of the cranial nerves III IV VI and the first branch of V produces partial or complete *internal or external ophthalmoplegia* impairment of the primary sensory modalities over the *forehead* and *corneal anesthesia* The signs and symptoms are ipsilateral unless the thrombosis spreads to the opposite cavernous sinus

Lateral Sinus Thrombosis —The lateral sinus is more frequently involved than the other venous sinuses because of the high incidence of *ear infections* (p 2145) Uncomplicated thrombosis of the lateral sinus produces *edema* and *tenderness* over the *mastoid area* with congestion and thickening of the peri auricular and jugular veins Infection of the thrombosed sinus brings about secondary infection of the brain or meninges with signs of *encephalitis brain abscess* or an *otogenic meningitis* (p 2148) Papilledema is a late sign

Gradenigo Syndrome —The Gradenigo syndrome consists of a palsy of

the external rectus (VI) and symptoms referable to the sensory division of V These structures are caught up with an inflammatory process near the apex of the petrous portion of the temporal bone (*petrositis*)

Treatment —The prognosis and treatment of sinus thrombosis have been altered by the introduction of the anti infective agents *Prophylactic chemotherapy* has reduced the incidence of these previously ominous complications to the point where they are now clinical rarities *active therapy* has all but abolished the necessity for performing formidable surgical procedures The addition of *anticoagulants* (p 1050) to the chemotherapeutic efforts holds even greater promise of effectual control

In the pre sulfonamide era there seemed no therapeutic approach to the problems of thromboses of the superior longitudinal and cavernous sinuses The operative treatment of thrombosis of the lateral sinus consisted in exenteration of the mastoid cells evacuation of the infected clot and ligation of the internal jugular veins The superimposition of an otogenic meningitis robbed the patient of any chance for recovery but even this complication responds extraordinarily well to intensive chemotherapy with sulfonamides (p 88) streptomycin (p 103) or penicillin (p 106)

ANEMIA OF THE CORD

There may be a rapid development of *paralysis* after profound systemic hemorrhage The suggested explanation is a resulting anemia of the spinal cord *Intermittent claudication* of spinal vessels with temporary level phenomena have also been described

ARTERIOSCLEROSIS OF THE CORD

Sclerotic changes of the vessels of the cord have been observed with secondary degeneration of the posterior and lateral columns and scattered lesions in white and gray matter These phenomena are difficult of diagnosis and must occur with great rarity

MENINGEAL HEMORRHAGE

Meningeal hemorrhages may be *epidural* or *subdural* They may produce immediate signs or manifestations due to the presence of the clotted blood or cicatrix

Meningeal Subarachnoid Hemorrhage —Subarachnoid bleeding follows trauma to the cord obstetric injuries convulsions hemorrhagic diathese spinal cord tumors or rupture of an aneurysm at the base of the brain (p 1444)

Subarachnoid hemorrhage in the region of the spinal cord is characterized by sharp *pain in the back* and symptoms referable to the nerve roots There may be *muscle spasm* from irritation or *paralyses* of the arm or leg The course and prognosis vary with the cause of the disturbance

Symptomatic relief is afforded by *lumbar puncture* which is not without risk and is not to be repeated if followed by evidences of more extensive damage

Pachymeningitis Interna Hemorrhagica and Adhesive Arachnitis —Organization of the meningeal clot leads to pachymeningitis interna hemorrhagica This affliction may give the manifestations of a *cord tumor* (p 1450) with compression phenomena (p 1433) The hemorrhage also may termi

nate in a *circumscribed serous meningitis* or an *adhesive arachnitis* whose symptoms and signs are those of a spinal cord neoplasm (p 1430)

Laminectomy and division of the adhesions or the evacuation of loculated fluid may clarify the entire clinical disturbance *Roentgen therapy* is said to be of additional value

OCCLUSION OF A SPINAL ARTERY

Occlusion of a spinal artery may result from cord tumors arteriosclerosis with thrombosis syphilitic endarteritis or embolization in the course of a septicemia

Spinal Cord Circulation —In the cervical region anterior and posterior spinal arteries arise from the vertebral arteries The *anterior spinal artery* is unpaired and has no anastomoses until it reaches the fifth cervical segment Below this level it is formed by the union of the lateral spinal arteries which arise from the intercostal and lumbar arteries The latter enter the spine along with the anterior roots The anterior spinal arteries on entering the cord give off lateral twigs which supply practically all of the spinal cord except the posterior columns

The *posterior spinal arteries* are two in number and run along the posterior surface of the cord *They receive no anastomoses until the fifth cervical segment Below this level they* are formed by the union of lateral spinal arteries that come also from the intercostal and lumbar arteries The posterior spinal arteries enter the spinal cord and supply the area of the posterior columns The *lateral spinal arteries* give off numerous anastomoses which supply and penetrate the whole periphery of the spinal cord

Clinical Manifestations —Compression of a lateral twig of the anterior spinal artery can produce an almost complete *Brown Sequard reaction pattern* (p 1476) except for the posterior column signs Severe compression of the anterior spinal artery before it branches may produce the syndrome of a *complete transection of the cord* (p 1457) except for retention of posterior column function Compression of the posterior spinal artery excludes both posterior columns (p 1470) leaving intact the remainder of cord function Compression of the peripheral anastomotic network of blood vessels may produce focal areas of necrosis in the periphery of the cord

Treatment —Thrombotic occlusions may be treated tentatively by *heparinization* as in cerebral thrombosis (p 1050)

HEMATOMYELIA

Hemorrhage into the spinal cord may be spontaneous or it may occur from trauma (p 14.0) cord tumors syringomyelia and blood dyscrasias (p 1085) The hemorrhage is primarily into the gray matter of the cord and often spreads up and down in the gray column Bleeding into the cord substance is destructive and may produce manifestations of partial or complete transection of the cord (p 1457)

Clinical Manifestations and Treatment —The outstanding signs and symptoms of hematomyelia are sudden focal *segmental pain and weakness* The cervical cord is most frequently involved with sudden pain in the shoulder or arm flaccid weakness of the hand and arm and segmental sensory signs of a syringomyelic type (p 150.) As a rule recovery is spontaneous and fairly complete over a period of six months although a high cervical hemorrhage is often fatal Treatment is symptomatic

CHAPTER 72

THE VOLUNTARY NERVOUS SYSTEM TRAUMA AND MECHANICAL DISTURBANCES

THE delicate nerve tissues of the brain and cord are well protected by the meningeal coverings the water bed' of the cerebrospinal fluid and the bony structure of the skull and vertebral column Significant injuries do not often occur but when they are encountered they have grave implications The practitioner who first sees the injuries to the nervous system is required promptly to recognize the extent of the trauma and exercise skilful judgment in the management of the situation until the expert neurosurgeon can be consulted Whether the injury involves brain or cord the first requirement is *protection against further damage* resulting from injudicious handling or overzealous attempts of therapy Later the control of *shock* (p 928) is attempted and efforts are made to reduce *increased pressure relationships within the skull or column*

HEAD INJURIES

Injury to the brain and its coverings may or may not be associated with fracture of the skull The latter in point of fact is of slight importance relative to the significance of damage to the underlying tissue The practitioner must not be too greatly impressed with positive radiological evidence nor should he be lulled into a sense of false security by a "negative report

SKULL FRACTURES

Skull fractures may be simple or compound linear depressed or penetrating

Linear Fracture —The simple linear break is most frequently seen and may be relatively unimportant though roentgenologically most impressive Most linear fractures occur at the *base* of the skull When the line of fracture crosses the middle fossa with rupture of the dura and ear drum bleeding occurs from one or both ears The fracture is then *compound* and carries the danger of *otogenic meningeal infection* (p 2148) If only the dura is torn the drum reveals a collection of blood clots in the middle ear, *paracentesis* at this time is ill advised Rupture of the dura also becomes obvious through *blackening of both eyes* or the escape of spinal fluid through the nose (*cerebrospinal rhinorrhea*) In the latter instance bacterial invasion of the meninges is invited through a *rhinogenic pathway* (p 2128)

Treatment —The uncomplicated linear fracture of the skull of itself requires no treatment beyond a period of careful observation for at least two to three weeks Compound fractures in which the meninges are exposed through the scalp nose or ear require intensive and immediate *prophylactic anti infective therapy* with *sulfonamides* (p 88) and/or *penicillin* (p 106) and *consultation* with the neurosurgeon With a *rhinorrhea* lumbar puncture is strictly avoided The temptation to evacuate blood clots from the middle ear also must be resisted

Depressed and Penetrating Fractures —Depressed fractures of the skull are often compound and usually lacerate the meninges or brain tissue The depressed fracture irrespective of the intracranial complications (p 1452) requires immediate *consultation* with the neurosurgeon who may deem it advisable to elevate the fragment as soon as the condition of the patient permits

Penetrating fractures produce laceration of the meninges and brain with the introduction of various foreign and infective material They require *surgical attention* as well as *supportive anti infective therapy* using the sulfonamides (p 88) and/or penicillin (p 106)

INTRACRANIAL INJURIES

Following a head injury the damages to the brain may or may not be related to the bony fracture The pathological manifestation may be limited to a *cerebral edema* (p 1451) with petechial hemorrhages or there may occur gross *contusion* or *laceration* (p 1452) with hemorrhagic phenomena such as *epidural subdural subarachnoid* and *subcortical bleedings* Superimposed on the manifestations due to anatomical changes are the syndromes of *shock* (p 928) and *increased intracranial pressure* (p 1421) Late sequels due to organization of clots include *pachymeningitis* (p 1448) *arachnitis* (p 1448) *encephalomalacia* and *cerebral atrophy*

CONCUSSION

Concussion is best illustrated by the knockout blow of the prize ring The pugilist momentarily *loses consciousness* and falls to the floor He is limp with dilated pupils apnea and an imperceptible pulse In less time than it takes to count ten he may rise to his feet and continue the bout but it is observed then that his coordination is poor his timing of blows loses its keenness and the knees seem to buckle After a complete knock out the patient may awaken with *headache* and may have an *amnesia* for the span of time that elapsed between the receipt of the blow and the awakening *the head is dulled dizziness is observed and an attempt to* rise to the feet is often associated with stumbling

Amazing recovery may supervene so that the defeated fighter makes his way to his dressing room takes off his clothes showers and dresses for the street without apparent ill effects On the morning after he may observe post concussive manifestations such as dullness headache dizziness nausea vomiting and mental retardation but experienced fighters in perfect condition maintain that there are no ill effects

Treatment —The ringside treatment of the knockout consists of sponging the face with water and the inhalation of *smelling salts* It would seem

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Linear Fracture —The simple linear break is most frequently seen and may be relatively unimportant though roentgenologically most impressive Most linear fractures occur at the *base* of the skull When the line of fracture crosses the middle fossa with rupture of the dura and ear drum bleeding occurs from one or both ears The fracture is then *compound* and carries the danger of *otogenic meningeal infection* (p 2148) If only the dura is torn the drum reveals a collection of blood clots in the middle ear *paracentesis* at this time is ill advised Rupture of the dura also becomes obvious through *blackening of both eyes* or the escape of spinal fluid through the nose (*cerebrospinal rhinorrhea*) In the latter instance bacterial invasion of the meninges is invited through a *rhinogenic pathway* (p 2128)

Treatment —The uncomplicated linear fracture of the skull of itself requires no treatment beyond a period of careful observation for at least two to three weeks Compound fractures in which the meninges are exposed through the scalp nose or ear require intensive and immediate *prophylactic anti infective therapy* with *sulfonamides* (p 88) and/or *penicillin* (p 106) and *consultation* with the neurosurgeon With a *rhinorrhea* lumbar puncture is strictly avoided The temptation to evacuate blood clots from the middle ear also must be resisted

Depressed and Penetrating Fractures —Depressed fractures of the skull are often compound and usually lacerate the meninges or brain tissue The depressed fracture irrespective of the intracranial complications (p 1452) requires immediate *consultation* with the neurosurgeon who may deem it advisable to elevate the fragment as soon as the condition of the patient permits

Penetrating fractures produce laceration of the meninges and brain with the introduction of various foreign and infective material They require *surgical attention* as well as *supportive anti infective therapy* using the sulfonamides (p 88) and/or penicillin (p 106)

INTRACRANIAL INJURIES

Following a head injury the damages to the brain may or may not be related to the bony fracture The pathological manifestation may be limited to a *cerebral edema* (p 1451) with petechial hemorrhages or there may occur gross *contusion* or *laceration* (p 1452) with hemorrhagic phenomena such as *epidural subdural subarachnoid* and *subcortical bleedings* Superimposed on the manifestations due to anatomical changes are the syndromes of *shock* (p 928) and *increased intracranial pressure* (p 1421) Late sequels due to organization of clots include *pachymeningitis* (p 1448) *arachnitis* (p 1448) *encephalomalacia* and *cerebral atrophy*

CONCUSSION

Concussion is best illustrated by the knockout blow of the prize ring The pugilist momentarily *loses consciousness* and falls to the floor He is limp with dilated pupils apnea and an imperceptible pulse In less time than it takes to count ten he may rise to his feet and continue the bout but it is observed then that his coordination is poor his timing of blows loses its keenness and the knees seem to buckle After a complete knock out the patient may awaken with *headache* and may have an *amnesia* for the span of time that elapsed between the receipt of the blow and the awakening the head is dulled dizziness is observed and an attempt to rise to the feet is often associated with stumbling

Amazing recovery may supervene so that the defeated fighter makes his way to his dressing room takes off his clothes showers and dresses for the street without apparent ill effects On the morning after he may observe post concussive manifestations such as dullness headache dizziness nausea vomiting and mental retardation but experienced fighters in perfect condition maintain that there are no ill effects

Treatment —The ringside treatment of the knockout consists of sponging the face with water and the inhalation of *smelling salts* It would seem

advisable to *compress the abdomen* at this time but experienced trainers and seconds prefer to loosen the tights and suspensories

The practitioner in the course of his routine duties rarely observes a brain concussion since the patient has already recovered by the time that he arrives It is his duty then, both from the standpoint of medical and medicolegal implications to insist upon an *observation period* of at least several days Accuracy of observation usually is seriously impaired by the administration of drugs particularly sedatives hypnotics and opiates as well as by the well intended efforts of bystanders who ply the victim with an alcoholic drink

A *radiograph of the skull* is mandatory since basilar linear fractures occur much more often and much more insidiously than is generally believed Furthermore at the time of the injury the attendant has no way of knowing whether or not the recovery represents a mere *free interval* to be followed by *deepening coma* (p 1295) within the course of the next several hours or days

In the home it should be possible to obtain hourly counts of the *respiratory and pulse rates* and four hourly rectal *temperature* readings Institutional observation requires additional estimations of systolic and diastolic *blood pressures* *Lumbar puncture* is postponed unless there are definitive localizing neurologic manifestations or evidences of increased intracranial pressure A neurosurgical consultation is imperative

The period of observation should be no less than seven to ten days if all is well This principle undoubtedly errs on the side of conservatism but it is far better to overtreat many patients than to be negligent of a single one at a price of chronic incapacitation and invalidism

CEREBRAL CONTUSION AND LACERATION

The transition from the syndrome of concussion to the phases of contusion and compression is difficult of recognition Under the latter circumstances the period of unconsciousness is prolonged the patient lies curled on one side and movement of the head is associated with sudden *explosive vomiting* Intense swelling of the brain and the exudation of lymph and blood elevate intracranial over venous pressure the cerebral veins collapse cerebral tissue becomes congested and the medullary centers suffer from ischemia Evidences of *increased intracranial pressure* (p 1471) are noted *respirations* slow and become irregular or of the Cheyne-Stokes variety a progressive *bradycardia* is observed in association with elevation of *systolic* and *pulse pressure* fluctuations in *temperature* from 1 to 8 degrees are noted in the absence of infection With *complete collapse* the blood pressure falls the pulse races breathing slows and the patient displays hyperpyrexia and manifestations of cardiac failure Mentally the patient varies between coma and restlessness there is frequent vomiting severe headache urinary and fecal incontinence or retention and a confusing leukocytosis Neurosurgical consultation is imperative

CRANIAL NERVE COMPLICATIONS

Together with the nonspecific factors of shock concussion and increased intracranial pressure a head injury may be accompanied by localizing symptoms referable to the cranial nerves *Anosmia* (I) is associated with

fractures which traverse the anterior fossa in the region of the cribriform plates The *eye grounds* (II) reveal important evidences of the extent of intracranial damage A retinal hyperemia occurs early with increasing engorgement a *papilledema* appears particularly in chronic subdural hematomas (p 1454) injury to the optic nerve is associated with *blindness* impingement on the chiasm produces a *bitemporal hemianopsia* When III IV or VI are involved the patient notes *diplopia* The sixth nerve is most frequently affected in basal fractures associated with hemorrhage Oculomotor paralysis (III) produces *external strabismus ptosis* and *dilatation of the pupils*

A tear of the infra orbital branches of V produces *anesthesia* along the nose and upper lip the upper teeth are involved when the dental branches are caught Injury to VII results in complete or incomplete *facial paralysis* and lesions of the auditory nerves (VIII) are revealed by *diminution in hearing* and *disturbances in equilibrium* The ninth tenth eleventh and twelfth nerves are well protected and are seldom damaged in head injuries

Treatment —The patient who has passed beyond the phase of concussion requires a *lumbar puncture* for purposes of diagnosis and therapy The presence of fresh blood indicates a subarachnoid hemorrhage (p 1445) clear fluid under increased pressure suggests edema or bleeding into brain tissues which do not communicate with the subarachnoid space

The suspicion of cerebral contusion or compression constitutes an urgent indication for *specialist consultation* Meanwhile the *conservative therapy* outlined in the management of concussion (p 1451) is continued but the practitioner errs if he fails to share the responsibility of the situation with his consultant

EPIDURAL HEMORRHAGE AND BLEEDING OF THE MIDDLE MENINGEAL ARTERY

A linear fracture which crosses the meningeal groove in the temporal bone may produce an epidural hemorrhage from a tear of the *middle meningeal artery* Under these circumstances a characteristic history is elicited *The patient* recovers consciousness and later lapses into *coma* The free interval varies from minutes to hours and may last several days It is characterized by *headache* and *tenderness* on the side of the injury There is often *contralateral motor weakness* due to the clot the first evidences are usually in the facial muscles and the pupil of the affected side usually is dilated

Signs of *increased intracranial pressure* (p 1421) accompany the second period of unconsciousness In contrast to concussion epidural hemorrhages are associated with *increased reflexes* on the contralateral side and positive pathological reflexes such as the *Babinski* The *spinal fluid* is under increased pressure and does not reveal blood unless there is additional oozing into the subarachnoid space *Surgical intervention* at this time with removal of the clot and control of bleeding may produce a phenomenally successful result

SUBDURAL HEMORRHAGE

Subdural hemorrhage results when the meningeal vessels are torn on the under surface of the dura The free interval may or may not be ob-

served the manifestations and treatment are similar to those of epidural hemorrhage (p 1453)

CHRONIC SUBDURAL HEMATOMA (PACHYMENINGITIS HEMORRHAGICA INTERNA TRAUMATICA)

The chronic subdural hematoma may follow a seemingly trivial blow The progress of the hemorrhage is so insidious that symptoms may not appear for weeks or months *Headache nausea vomiting disturbances in vision papilledema restlessness negativism* and *untidiness* are noted These often are interpreted as evidences of *traumatic neurosis* (p 1356) but this diagnosis cannot be maintained in the presence of localizing signs such as Jacksonian *convulsions changes in the reflexes alterations in the visual fields increased spinal fluid pressure* or *xanthochromia*

As in the instance of the epidural hemorrhage the chronic subdural collections are amenable to *surgical therapy*

SUBDURAL HYGROMA

After head injuries the ruptured arachnoid membrane may permit the cerebrospinal fluid to escape into the subdural space The material is not absorbed and forms a foreign body similar to the subdural hematoma (p 1454) The findings are identical until exploration has been accomplished

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage which is the result of trauma may be so extensive as to cover most of a cerebral hemisphere The blood seeps down over the cord and the spinal fluid reveals evidences of fresh bleeding

The symptoms and signs of subarachnoid hemorrhage from trauma are identical with those that occur from rupture of an arteriosclerotic vessel (p 1438) or an aneurysm (p 1444) The patient has *severe headache* and may be in complete *collapse* Stiffness of the neck on flexion a positive Kernig sign leukocytosis bloody spinal fluid and occasional localizing symptoms are observed

The problems of *therapy* particularly referable to the advisability of lumbar puncture are elsewhere discussed (p 1446)

SUBCORTICAL HEMORRHAGE

Subcortical hemorrhages usually occur at the juncture of gray and white matter If they involve the motor area or the internal capsule they may produce a *contralateral alternating hemiplegia* In the midbrain or brain stem they result in the characteristic picture of a *decerebrate rigidity* the entire body is stiff the arms are extended and rotated inward the wrists are flexed the legs are rigid and extended with pronation of the feet (slipper position) *Treatment* is expectant

TRAUMATIC PNEUMOCEPHALUS

Following trauma to the skull a communication may be established between one of the paranasal or mastoid sinuses and the cranial cavity A portal of entrance is created which permits air to enter the ventricles subarachnoid or subdural spaces or even penetrate into brain substance The condition is suspected when the patient notes *violent headaches* fol

lowing coughing sneezing or blowing of the nose A cerebrospinal rhinor rhea is frequently present Roentgenograms clearly depict the condition which requires prompt and intensive *chemotherapy* often followed by *operative interference*

POSTTRAUMATIC DISTURBANCES

Following the head injury a variety of persistent symptoms may be encountered At times these are continuations of the original complaint but again they may not arise for several weeks or months The principal features are vague disturbances such as headaches dizziness weakness ir ritability changes in memory and disposition insomnia or anxiety Less often localizing phenomena are seen such as posttraumatic epilepsy with Jacksonian attacks evidences of compression of cranial nerves (p 1480) or the features of a pachymeningitis hemorrhagica interna (p 1454)

The posttraumatic sequels are usually suspected of being of functional origin as in a *traumatic or litigious neurosis* (p 1356) The differential diag nosis is most difficult but the presence of any organic finding in the physical examination or any positive manifestation in the spinal fluid requires the practitioner to abandon any thought that the complaints are on anything other than a real and tangible basis *Specialist consultation* is mandatory since medicolegal complications are the rule rather than the exception

ARTERIOVENOUS ANEURYSM

With fracture of the *sphenoids* an abnormal opening may be estab lished between the wall of the *internal carotid artery* and the *cavernous sinus* The back flow results in *stabbing pain* centered behind the affected eyeball the production of a *pulsating exophthalmos* with an *audible bruit* dilatation of the veins of the upper lid forehead and temple papilledema engorgement of the scleral vessels and disturbances in vision

Specialist consultation is required to consider the possibility of gradual occlusion of the involved carotid

BIRTH PALSIES (LITTLE S DISEASE CEREBRAL DIPLEGIA IN FANTILE CEREBRAL PARALYSIS CEREBROCEREBELLAR DIPLEGIA AND INFANTILE SPASTIC HEMIPLEGIA)

The birth or cerebral palsies are probably due to injury of brain tissue as the result of *parturition* Some may be the result of infantile disturb ances in *blood coagulation cerebral hypoplasia* or *degeneration* but the end results do not differ

Clinical Manifestations —The usual clinical manifestation of cerebral palsy is *spastic diplegia* or Little s disease The infant is observed to hold its legs in adduction As the child begins to walk the gait appears jerky the youngster walks on his toes the thighs are adducted the knees rub together and the legs cross (*scissors gait*) Similar changes may be observed in the arms The deep reflexes are lively the Babinski and confirmatory signs are elicited and the abdominal and cremasteric reflexes are generally absent The spastic diplegia often is associated with imbecility epileptic or Jacksonian convulsions cranial nerve palsies (p 1480) dysarthria abnormal involuntary and associated movements of a choreo athetotic variety

On suspicion of a cerebral palsy, the *neurosurgeon* is summoned and spinal fluid examination is indicated With a bloody tap surgical exploration is considered since later procedures hold little promise

Treatment —Many spastic paraplegics make remarkable accommodations to their affliction With patience, muscle reeducation and the execution of orthopedic procedures the handicapped child may lead a useful and reasonably complete existence Best results are obtained in institutions which have specially trained staffs

There has been some recent encouragement from the use of intocostrin a preparation allied to *curare* for the relief of spasm An intramuscular dose of 1 cc per 20 lbs of body weight but not to exceed 4 cc is followed in a few hours by amazing relaxation which may last two or three days after which another injection may be given See p 3888

INJURY TO THE SPINAL CORD

Injuries to the spinal cord may or may not be associated with damage to the vertebrae As in the instance of head injury there may be concussion contusion penetration or compression of the cord with or without bleeding into the meninges

SPINAL CONCUSSION OR SHOCK

Spinal concussion is as genuine a clinical entity as concussion of the brain (p 1451) The syndrome follows jarring injuries with or without a solution of continuity In all likelihood the local pathologic lesion consist of a focal edema of the cord with punctate hemorrhages

Clinical Manifestations —In spinal shock there is loss of motor sensory and reflex action but maintenance of the control of the sphincters Spinal shock may be marked by a clearly delineated *level* as in spinal cord tumors (p 1433) The *zone of anesthesia* may be surmounted by a *girdle of hyperesthesia* The paralyzed limbs are *flaccid* and may remain inert for ten days to two weeks At the end of this time a phase of *reflex hyperexcitability* may be encountered, a Babinski sign is elicited the thigh and knee are flexed the ankle is extended and trophic changes may appear in the skin with the formation of decubitus ulcers

The terminal phase of spinal shock is that of *diminished reflex excitability* with retention of urine secondary bladder infection and an ascending uropathy (p 2334) Bedsores appear and the paralyzed muscles exhibit atrophy and wasting

Treatment —The management of spinal cord shock calls for *expert consultation* Overzealous therapy may inflict irreparable damage and operative interference holds little promise The management of the *cord bladder* is the most urgent problem and is elsewhere discussed (p 2331)

CONTUSIONS AND LACERATIONS OF THE CORD

The clinical manifestations of contusion and laceration of the cord differ very little from those of cord concussion There may be more clearly defined localization of the injury and the finding of blood in the cerebrospinal fluid The differentiation is of academic importance since little can be done by way of therapy

CORD COMPRESSION

Cord compression may be the result of the direct impingement of a neoplasm a blood clot or a fracture dislocation the encroachment upon the intravertebral space of edema inflammatory exudate or a vascular swelling

The symptoms and signs of cord compression are described elsewhere (p 1430) If the symptoms warrant *laminectomy* is advisable for diagnostic and therapeutic purposes If the mechanisms producing the compression cannot be removed a decompression is accomplished

TRANSECTION OF THE CORD

Transection of the cord may result from trauma or inflammation (*transverse myelitis*) In either instance identical manifestations are noted

Clinical Manifestations—Spinal cord transection results in a *flaccid paralysis* below the level of the lesion muscle tone is lost the limbs are cold and cyanotic there is absence of reflexes and sensation the sphincters of the bladder and rectum are paralyzed As the result of involvement of the tracts of the involuntary nervous system there is usually a temporary drop in blood pressure which may last from twelve to twenty four hours

In the beginning transection of the cord does not differ in its manifestations from *spinal concussion* (p 1456) However at the end of three weeks few changes are noted in the organic findings the phase of hyper excitability is not observed and there is no return of sensation or muscle power

Treatment—The spinal cord transection is a serious lesion Particular care is required in transportation if the injury is thoracolumbar the *patient is best carried prone on a well-padded litter* with cervical lesions the patient is carried supine Skeletal traction with tongs applied to the skull may be tried in the effort to reduce cervical fractures

Surgery—As soon as the patient is hospitalized specialist consultation is required to discuss the feasibility of performing a *closed reduction* of a fracture or dislocation or a *palliative laminectomy* over the level of the lesion The urgency of a decision is emphasized by the fact that failure to relieve existing pressure on the cord within eighteen hours is followed by permanent damage

While there is considerable difference of opinion concerning operative procedure most surgeons agree that laminectomy is indicated only when the neurological signs are progressive The operation should not be performed merely because there is radiographic evidence of bone pressure or the demonstration of a partial or complete block by the Queckenstedt test (p 1434)

Conservative Therapy—The later conservative management of cord transection requires unusual medical and nursing skill The treatment of the *paralyzed bladder* is a subject of considerable difference of opinion Tidal drainage can be used for weeks but in permanent lesions it is necessary to do a suprapubic cystostomy with prophylactic and continuous *chemotherapy* using the sulfonamides Repeated catheterizations and the use of an indwelling catheter invite infection and augment the difficulties of medical care Later in the course of the affliction the cystostomy tube

may be clamped off and the patient makes effort at voluntary urination employing compression in the hypogastric region If this is successful the tube is withdrawn

The problem of *defecation* is best managed by a daily enema or irrigation *Hypostatic pneumonitis* is avoided by frequent changes in position and the effects of the sulfonamide drugs used for the urinary tract condition A *cradle* is placed over the lower limbs which are *splinted* to prevent deformities from foot drop *Light massage* is not objectionable *Re educational exercises* under expert tutelage, are advised for the limbs as soon as motor activity begins to return Pressure points are protected by padding and the frequent application of talcum powder After the initial lumbar puncture for diagnostic purposes further drainages of cerebrospinal fluid are hazardous

HEMORRHAGIC LESIONS OF THE SPINAL MENINGES

As in the instance of the cranial meninges the spinal cord coverings may become irritated or compressed by fluid or clotted blood A *subdural hematoma* acts in the manner of a *spinal cord neoplasm* (p 1430) Organ ization of the clot produces the manifestations of a *pachymeningitis in terna hemorrhagica* (p 1454) or a *diffuse arachnitis* (p 1448) These mani festations become apparent only upon *exploratory laminectomy* Under these circumstances simple removal of the abnormal tissue or division of adhesions may result in the relief of the clinical abnormalities

FRACTURES AND DISLOCATIONS OF THE SPINE

Fractures and dislocations of the spine assume importance only in so far as they affect the underlying meninges or nerve tissues *Compression fractures* result from *indirect violence* transmitted through the feet head or shoulders or by sudden marked extension or flexion of the body as in an accident or a therapeutically induced *convulsion* (p 1330) The fracture may be primary or secondary to a malignant metastatic or a chronic in flammatory granuloma

Fracture dislocations of the spine involve a break of the body of the vertebra or its processes and associated dislocation of the transverse processes This injury in addition to compression of the spinal root causes *cord compression* and vascular disturbances leading to a *hematomyelia* (p 1457) It requires specialist consultation

INJURIES TO THE PERIPHERAL NERVES

Injuries to the peripheral nerves assume great importance since their effects which may be devastating are amenable to prompt and competent nerve surgery

Etiology—The peripheral nerves of the extremities are particularly vulnerable to traumatic disturbances They may be severed or crushed by direct injury with gunshot or stabbing they may become involved in dislocations and fractures they may be caught up in a callus formation or suffer injury from the pressure of a cast or bandage they may suffer compression from trauma in sleep under anesthesia during a prolonged intravenous injection or as the result of an awkward position during a surgical procedure they may be squeezed or stretched by a neon or

an osteo arthrotic spur they are crushed by the pressure of the scalenus anticus with or without the presence of a cervical rib they may be injured at birth or by spinal caudal or epicaudal anesthesia through accidental injection into the nerve or through the toxemia of a systemic disease such as diphtheria botulism or leprosy

Clinical Manifestations —Injury to a peripheral nerve is associated with phenomena of irritation or paralysis The former causes *paresthesias causalgia* (p 1476) and *neuralgia* (p 1490) With paralysis there is loss of touch temperature pain and deep sensibility in the area supplied

Motor irritations result in spasms tics and fibrillations while *paralysis* produces a reaction of degeneration to electrical stimulation with loss of reflex loss of strength flaccidity and diminution in volume Trophic vasomotor and secretory changes accompany peripheral nerve injuries The skin becomes glossy and smooth changes are noted in sweating and the appearance of the nails and hair ulcerations are prone to develop and persist

The localization of the injured peripheral nerve is discussed in greater detail in the charts that accompany the discussion of the *peripheral mononeuropathies* (p 1490)

Treatment —The establishment of a definitive peripheral nerve lesion is an urgent indication for *specialist consultation* The specialist with great dexterity is often enabled to accomplish *primary or secondary suture* Immediate repair is favored unless there are evidences of infection or contamination in the near vicinity Other useful operative procedures include *neurolysis* in which the nerve is dissected free from scar tissue and placed in a new bed division of the scalenus anticus removal of the nucleus pulposus or excision of a neoplasm

CHAPTER 73

THE VOLUNTARY NERVOUS SYSTEM INFECTIONS

Elective Localization of Neurologic Infections
Nonsuppurative Encephalomyelomeningitides
  Tuberculous Meningitis
  Tabes Dorsalis
  Meningovascular Syphilis
Purulent Encephalomyelomeningitides

BACTERIAL infections of the voluntary nervous system usually represent secondary localizations following systemic invasions With demonstrable organisms such as the meningococcus an *initial bacteremia* is recognized and this phase may or may not be followed by a meningeal reaction In the instance of the more frequent virus infections the sequence of events is not capable of proof but may be deduced from the clinical course in which the neurologic phenomena follow the systemic manifestations

The *tissue reactions* to infection are a variable *Coccal invasions* are associated with suppuration but most other reactions are concerned with lymphocytic and fixed tissue cell responses Infrequently, the structures are damaged by *circulating toxins* but most often the disturbance results from the presence of *bacterial bodies*

ELECTIVE LOCALIZATION OF NEUROLOGIC INFECTION

The majority of bacterial invaders exhibit *specific affinities* for constituent portions of the voluntary nervous system The exceptions are the organisms responsible for *syphilis* and *tuberculosis* The former variously attacks the brain (*general paresis*) cord (*tabes dorsalis*) meninges or blood vessels (*meningovascular lues*) and may additionally react by *gumma formation* in any conceivable site The tubercle bacillus has the potential for the production of *meningitis* (p 1462) *miliary tubercles* or *tuberculomas*

With these exceptions the bacterial invaders of the voluntary nervous system appear to be more discriminating in their localizations The toxins of *diphtheria* and *tetanus* specifically attack the *peripheral nerves* and the *myoneural junctions,* the viruses of *zoster* and *acute anterior poliomyelitis* localize in peripheral nerves or the cord the *pyogens* set up *purulent meningitis* (p 1462) and the majority of the *encephalitides* result from viral infections

NONSUPPURATIVE ENCEPHALOMYELOMENINGITIDES

Nonsuppurative infections of the brain cord peripheral nerves and meninges without exception are local manifestations of systemic invasions

Etiology—Of the roster of living invaders listed in the table of Elective

Localization of Infection in Nerve Tissue (p 1462) only coccal invaders and *H influenzae* produce suppurative inflammation The remaining cocci bacilli spirochetes viruses and protozoa evoke a lymphocytic or fixed cell response

Clinical Manifestations—The clinical manifestations of the non sup purative encephalomyelomeningitides are considered in the Section de voted to the Infections (p 442) The three notable exceptions are tabes dorsalis meningovascular syphilis and tuberculous meningitis which for

TABLE 93—INFECTIONS INVOLVING THE SPINAL CORD AND PERIPHERAL NERVES

Specific Infection	Clinical Characteristics	Diagnostic Criteria
Diphtheria (p 309)	Membranous inflammation of naso-pharynx with secondary peripheral neuritis often bulbar in distribution	Culture of local mem brane specific anti toxin therapy
Tetanus (p 294)	Infected wound with secondary toxemia sensitizing myoneural junctions and causing tetanic and convulsive phe-nomena	Culture of wound spe-cific antitoxin therapy
Leprosy (p 273)	Peripheral thickening of nerves espe-cially ulna with anesthetic nodules	Stains of nasopharyngeal secretion for organism
Syphilis (p 331)	Gumma of cord rarely seen manifesta tions of cord tumor with positive serology	Blood and spinal fluid serology positive ther apeutic response to iodide
	Tabes dorsalis (locomotor ataxia) (see p 1464)	Blood and spinal fluid serology tabetic gold curve
Herpes Zoster (p 435)	Shingles vesicles and pain along course of peripheral nerve	Clinical appearance
Acute Anterior Poliomyeli tis (p 457)	Generalized infect on with secondary flaccid paralysis of bulbar regions or extremities ascending paralysis (Lan dry)	History of endemic cl ar spinal fluid with nor mal sugar content early polymorphonuclear pleocytosis followed by lymphocytosis
Pyogenic Organisms	Cord abscesses causing reaction pat-tern of cord neoplasm (see p 1430)	Operative findings

purposes of convenience are presented despite the inconsistency in the present chapter

Differential Diagnosis—The differential diagnosis of the nonsuppurative encephalomyelomeningitides has been presented elsewhere in tabular form (p 449) Main reliance is placed upon epidemiology the clinical history and the more objective information gleaned from examinations of the cerebrospinal fluid and of the serum for virus neutralizing bodies and for fixation of complement

Treatment—The treatment of nonsuppurative inflammation of nerve tissue is a most disappointing experience Exceptions to this generality are

happily found in the prevention of rabies and in anti syphilitic measures in tabes dorsalis

TUBERCULOUS MENINGITIS

Tuberculous meningitis is usually a terminal manifestation of an acute miliary tuberculosis in childhood and infancy In older individuals or adults, contamination of cerebrospinal fluid may occur long after the hematogenous seeding and may appear as the only active manifestation of a tuberculous infection

TABLE 94 —INFECTIONS INVOLVING THE MENINGES

Specific Infection	Clinical Characteristics	Diagnostic Criteria
Staphylococcic Meningitis Streptococcic Meningitis	Secondary to compound skull fracture otogenic (p 2148) or rhinogenic (p 2128)	Purulent fluid with gram positive cocci
Pneumococcic Meningitis	As above but also may be secondary to bacteremia	Purulent fluid, with gram-positive cocci
Meningococcus Meningitis (p 213)	Meningeal reaction pattern with acute or chronic bacteremia	Purulent fluid with gram negative cocci
Influenzal Meningitis (p 286)	Meningeal reaction pattern following respiratory infection or bacteremia	Purulent fluid with gram negative bacillus
Tuberculous Meningitis (p 1462)	Meningeal reaction pattern usually in tuberculous children Choroid tubercles often seen	Clear lymphocytic spinal fluid with coagulum containing acid fast bacilli
Syphilis (p 1468)	Meningovascular type produces meningeal reaction pattern in luetic	Positive serology in blood and clear spinal fluid good response to anti luetic treatment
Epidemic Parotitis (p 483)	Meningeal reaction pattern following mumps	Clear lymphocytic fluid
Lymphocytic Choriomeningitis (p 448)	Meningeal reaction pattern following grippal attack	Clear or ground glass spinal fluid with mononucleosis Virus neutralizing bodies appear later

Clinical Manifestations —Tuberculous meningitis may start insidiously or acutely In the infant it is noted that the child has become irritable constipated and drowsy appetite fails and food is vomited seemingly without cause At first the temperature variations may be mild and variable but later fever increases until the terminal hyperpyrexia With progression of the lesion the fontanelle bulges drowsiness proceeds to the point of stupor convulsions are interpolated and evidences of increased intracranial pressure become manifest The retinal veins appear full the pulse rate alternates between bradycardia and tachycardia and the respira

TABLE 9 —INFECTIONS INVOLVING THE BRAIN

Specific Infection	Clinical Characteristics	Diagnostic Criteria
Rheumatic Fever (p 190)	Chorea	Clinical sequence
Pertussis (p 2 8)	Encephalopathy after whooping cough	Clinical sequence
Tuberculosis (p 252)	Tuberculoma simulating brain tumor (p 1419)	Other evidences of tuberculosis or operative findings
Brucellosis	May produce encephalitis or meningitis (p 314)	Isolation of organism or positive skin test
Syphilis (p 331)	Gumma simulates brain tumor (p 1419)	Response to iodide
	Meningovascular lues	Positive serology and response to antiluetic therapy
	General paresis (p 13)	Positive serology paretic gold curve
Measles (p 409)	Encephalitis (p 44) after eruptive disease	Clinical sequence
Rubella (p 41)	Encephalitis (p 44?) after German measles	Clinical sequence
Varicella (p 4 0)	Encephalitis (p 44) after chickenpox	Clinical sequence
Variola (p 4 4)	Encephalitis (p 44) after smallpox	Clinical sequence
Vaccinia (p 4 8)	Encephalitis (p 44) after vaccination	Clinical sequence
Epidemic Encephalitis (p 441)	Multiple involvements in epidemic form	Clear spinal fluid with moderate lymphocytosis
St Louis Encephalitis (p 454)	As above	Virus neutralizing bodies in convalescence
Equine Encephalomyelitis (p 4 1)	Cerebral and cord involvements	Ground glass fluid with lymphocytosis and virus neutralizing bodies in convalescence
Japanese B Encephalitis (p 44?)	Probably corresponds to American types of encephalitis	
Russian Seasonal Encephalitis	Probably corresponds to American types of encephalitis	
Rabies (p 439)	Hydrophobia following bite	Examination of brain of dead animal
Torulosis (p 496)	Encephalitis or meningeal reaction pattern	Torula in spinal fluid
Amebiasis (p 523)	Brain abscess in dysentery patient	Ameba in stool or abscess pus
Trypanosomiasis (p 531)	African sleeping sickness	Organisms in blood smears

tory rate may slow speed or become totally irregular Older patients complain of headache and changes in personality are noted

Physical examination reveals the bulging fontanelle in infancy stiffness of the neck opisthotonos positive Kernig and Brudzinski signs and finally the choroidal tubercle reveals itself by ophthalmoscopy

Laboratory Findings —The finding of tubercle bacilli in the cerebrospinal fluid leaves no doubt as to the diagnosis of tuberculous meningitis The fluid is under increased pressure and may appear clear or somewhat hazy The globulin content is increased but sugar and chloride contents are reduced The cell count is elevated to several hundred primarily lymphocytes If the fluid is permitted to stand over night a pellicle forms When this is picked out and stained by the carbolfuchsin method (p 52)

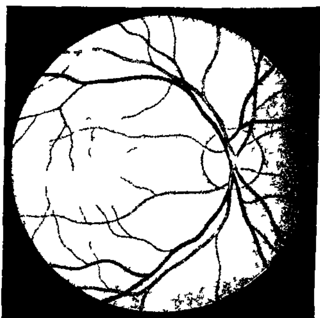


Fig 276 —Miliary tuberculosis of the choroid The yellowish gray color of the spots is due to edema and opacity of the overlying retina (Koellner)

acid fast organisms can be demonstrated without exception if the search is conducted with patience for several hours

Treatment —Tuberculous meningitis is a universally fatal disease despite all efforts and heroic therapy If the physician is impelled to employ measures of desperation he may try massive doses of streptomycin intravenously and intrathecally (p 106) in association with promin used orally (p 267)

TABES DORSALIS (LOCOMOTOR ATAXIA)

Tabes dorsalis represents a *syphilitic infection* in which there is a diffuse and bilateral involvement of the *posterior horn cell group* with primary or secondary degeneration of the *posterior columns* Additionally there may be lesions of the *ascending spinal cerebellar pathways* and the cranial nerves particularly II III IV and VI

Clinical Manifestations—The initial symptoms of tabes dorsalis consist of *painful dysesthesias* from irritations of the affected posterior roots With impairment of the centers for vibration position and deep pressure the patient is disoriented relative to his position in space and notes difficulty while walking in the dark or sways when he covers his face with his hands while washing Examination at this time reveals a *loss of vibratory sense* a *positive Romberg test* diminution or complete *loss of deep reflexes* in the knee and ankle regions *absence of pain sense* on compression of the Achilles tendon or the testes and *patches of anesthesia* along the lateral sides of the calf thigh or foot

Ocular Disturbances—Almost without exception there are accompanying ocular manifestations The *pupils* are irregular in size and shape and unequal (*anisocoria*) the light reflex is lost but the power of accommodation is retained (*Argyll Robertson pupil*) There may be weaknesses or paralyses of the external ocular muscles and a *primary optic atrophy*

Locomotor Ataxia—As the sensory manifestations progress the patient reaches the ataxic stage where he is unable to walk with any degree of steadiness Characteristic gait changes appear in which the foot is raised high off the ground wavers unsteadily and then is flapped noisily to the floor The mechanical damage produced by this method of locomotion leads to the formation of the *Charcot joint* (p 2953) The weight bearing structures of the hip knee ankle and spine are most often involved in a *painless deforming arthropathy* with such complete destruction of the joint structures that the end stage is a bag of bones

Tabetic Crises—The most horrible of the tabetic symptoms are the crises which may be *peripheral* or *visceral* Agonizing lightning pains occur in the muscles of the extremities abdomen or chest They are described by the patient as burning gnawing lancinating twitching or resembling a stabbing with a hot knife The attacks come on with the rapidity of lightning they may last for several hours or days with brief intervals of freedom Other associated abnormal phenomena include paresthesias numbness sensations of cold warmth and tingling

The visceral crises give rise to many difficulties in diagnosis They may be *gastric salivary lacrimal rectal vesical laryngeal pharyngeal* or *sexual* The most frequent is the *gastric crisis* characterized by recurrent abdominal distress vomiting and pain resembling an organic *intra abdominal affliction* (p 3555) the referred phenomena of a *coronary occlusion* (p 983) or an *angina pectoris* (p 890)

Disorders of the Sphincters—The progression of the tabetic process eventually leads to involvement of the sphincters of the bladder and rectum Either *incontinence* or *retention* may be observed Under the latter circumstance the bladder eventually becomes distended with the resultant introduction of infection and the possibility of an *ascending urinary sepsis* (p 2334) An early complaint is *dribbling* with difficulties in the initiation of the urinary stream and a feeling of incomplete emptying after micturition At the time of sphincteric involvement *sexual debility* and eventual *impotence* are almost invariable though the latter may occur despite the integrity of the mechanisms of urination and defecation

Trophic Disturbances—Local trophic disturbances occur in the nature of *atrophies* and *penetrating ulcers* Particularly in those with gastric cri

ses and disturbances of bladder function a *generalized cachexia* develops progressively and the patient loses weight and strength in the manner of one suffering from a carcinomatosis (p 572)

Laboratory Findings—The laboratory findings in tabes dorsalis may be confirmatory or wholly confusing If they are characteristic the fluid is clear, there may be up to 100 *lymphocytes* in the cell count blood and spinal fluid *serologic reactions* are positive, the *colloidal gold test* shows little or no change in the first two tubes moderate alteration in the third fourth fifth, sixth and seventh tubes and a normal appearance thereafter Such a reading might be 0123332100

In the *early stages of syphilis* primary as well as secondary it is not unusual to obtain the laboratory findings that are characteristic of a tabes without any confirmatory clinical manifestations Asymptomatic cerebro spinal syphilis is much more important of recognition than the fully developed tabetic syndrome since therapy, at this time holds great promise of cure' In the later stages of a *burned out'* tabes at the height of the organic neurologic findings the laboratory data may be completely misleading The serologic tests of the blood and spinal fluid may be normal even when larger quantities are used and the colloidal gold test reads zero in all dilutions The clinician is required to establish the presence of the tabetic involvement by his physical findings despite the denials of the laboratory tests

Course—The course of tabes dorsalis is variable The more obvious and blatant manifestations are those in which there is a progressive advance toward invalidism and a fatal issue The comparative rarity of tabes dorsalis in proportion to the number of positive spinal fluid findings in infectious syphilis indicates that most patients pursue a more favorable course An appreciation of these facts is required in the evaluation of therapeutic procedures

Treatment—The treatment of tabes dorsalis is individualized and is dependent upon the stage of the disease and the presenting symptoms

The introduction of *penicillin* in the treatment of symptomatic and asymptomatic tabes holds high promise This prospect contrasts with prior efforts of which Wechsler stated that he had 'never seen a case of tabes cured nor a disappearance of signs once they had become fixed

While it is too soon to assess final results or enunciate an optimum treatment schedule we favor a course of penicillin injections given intramuscularly at three hour intervals for eight to ten days until 10 000 000 units have been delivered We advocate repetition of the course at intervals of six months until clinical and serologic control is absolute

For the private practitioner we recommend no complicating endeavors We oppose intrathecal therapy with penicillin as with the arsenicals We reserve adjuvant arsenotherapy for those who are penicillin resistant and in whom the risks of treatment are justified Thus far though it is too soon to be dogmatic it appears that penicillin therapy of tabes holds high promise for clinical if not serologic control without *appreciable* added benefits from concomitant arsenotherapy

The patient must be warned of the guarded prognosis and of the necessity for regular reexaminations of blood and spinal fluids He must realize that retreatments at three or six month intervals are *imperative*

TRYPARSAMIDE—The advisability of using *tryparsamide* arises sooner or later in the treatment of each tabetic Those who advocate injections of this drug ordinarily give intravenous injections of 30 to 50 mg per kg (2 2 pounds) of body weight An average adult dose approximates 3 gm dissolved in 10 cc of sterile distilled water Weekly injections are given in courses of 8 to 16 followed by rest periods of approximately six weeks Many clinicians including the present authors have little faith in trypar samide and much fear concerning its toxicity *Untoward manifestations* include tinnitus dizziness nausea vomiting headache urticaria dermati tis jaundice and visual disturbances that may progress to optic atrophy Against these risks there are few positive and convincing evidences of improvement The patient may report gain in weight and a sense of well being but there are no significant changes in the objective findings If the practitioner is driven to its use in desperation he should insist upon pre liminary expert consultation in order to share the onus of toxicologic disturbances

Hyperthermia—The favorable effects of fever treatment in *general paresis* (p 1377) stimulated efforts to apply similar technics for the relief of tabes Unfortunately the same successes have not been experienced and most critical observers including the author reserve hyperthermia for patients whose progress is unsatisfactory despite best efforts with chemotherapy

Control of Pain—The tabetic in addition to antiluetic therapy re quires palliation particularly relative to the *tabetic crises* For the relief of these agonizing pains many therapeutic endeavors have been instituted and their numbers testify to their relative uselessness Wechsler reports that he has seen no good results from the use of galvanism or faradism albeit they are recommended He states that diathermy may on occa sions give relief to pain and so may x ray therapy (p 3796) This has not been our experience and our only successes are with the daily intra venous injections of large doses of *thiamine chloride* (p 622) Given in amounts of 100 to 200 mg we have had surprisingly satisfactory results when all other measures have failed

Symptomatic *drug therapy* in the tabetic is attended with consider able risk of addiction (p 3815) It is not only the lightning pain but the fear of the onset of these anguishes that lead the tabetic to use increas ingly greater doses of the *analgesics hypnotics sedatives* and *opiates* Many become *chronic alcoholics* in their understandable efforts to secure surcease All else failing localized pain may be subjected to surgical ther apy by *paravertebral injections chordotomy* or *sectioning of the poste rior roots*

Orthopedic Treatment—*Reeducation methods* give excellent results in teaching the tabetic to control his unruly legs *Braces* and *supports* are used to overcome the hyperextensibility of the joints which precedes the tabetic arthropathy

Treatment of Urinary Difficulties—The attention of the therapist often is directed to the problem of dealing with the urinary bladder In the face of urinary retention and dilatation of the bladder it is our belief that optimal management requires the institution of a permanent *supra pubic cystostomy* with fortification by *prophylactic chemotherapy* using

the sulfonamides Repeated catheterization besides being a nuisance invites urinary sepsis (p 2334)

MENINGOVASCULAR SYPHILIS

In addition to the definitive entities of general paresis (p 1377) and tabes dorsalis (p 1464), syphilis may evoke protean neurologic manifestations due to the production of a meningeal reaction or to involvement of nerve tissue resulting from vascular injury and gumma formation The clinical manifestations are capable of simulating any of the organic diseases of the central nervous system, and the diagnosis is not established until the laboratory findings have been completed In the presence of a positive Wassermann test on blood or spinal fluid of spinal pleocytosis or changes in the colloidal gold reaction the practitioner is entitled to consider, but not definitely to commit himself to the diagnosis of syphilis as the etiologic agent Until the completion of the therapeutic test accomplished by intensive penicillin therapy, hyperthermia (p 3789) and supplementation by iodides for dissolution of the gumma (p 608) there is always the possibility that the syphilitic just as his non infected fellow may be afflicted with an unrelated neurologic disorder such as a brain tumor, meningococcus meningitis or multiple sclerosis for example

PURULENT ENCEPHALOMYELOMENINGITIS

Suppurative inflammations involving brain cord or meninges may be the result of direct extension of pyogenic processes or they may be metastatic in connection with pyemia or amebic dysentery

EXTRADURAL AND SUBDURAL ABSCESS

Subdural and extradural brain abscesses arise as complications of inflammatory processes involving the *skull* the *nasal accessory sinuses* the *middle ear* the *mastoid* or the *lateral superior longitudinal* or *cavernous venous sinuses* (p 1446)

Clinical Manifestations —The complication of brain abscess is usually clouded by the clinical manifestations of the more fundamental process The suspicions of the practitioner are aroused when the systemic manifestations seem out of proportion to the extent of the local process when additional disturbances point to increased intracranial pressure or focal neurological involvements

The signs of *increased intracranial pressure* (p 1421) include headache skull tenderness persistent nausea projectile vomiting congestion of the retinal veins and papilledema *Localizing neurologic signs* are dependent upon the site of the suppuration With *lateral sinus infections* (p 1446) there are irritations of the fifth sixth and eighth cranial nerves as shown by ophthalmoplegia diplopia facial anesthesias or paralysis tinnitus and disturbances in hearing *Frontal lobe abscesses* (p 1469) associated with infections of the orbit and the nasal accessory sinuses are less clearly delineated The patient may reveal subtle changes in disposition delirium or convulsions

Treatment —The approach to the treatment of extradural or subdural abscess involves an attack on the more fundamental lesion Thus in the commonest variety associated with mastoiditis and thrombophlebitis of

the lateral sinus therapy with sulfonamide (p 88) streptomycin (p 103) or penicillin (p 106) is combined with exenteration of the infected bone cells removal of the infected clot ligation of the jugular vein and drainage of the abscessed area in the brain Optimal management requires the cooperation of otologist and neurological surgeon

BRAIN ABSCESS

Brain abscess within the substance of the cerebral hemispheres is most often metastatic It follows *pulmonary suppuration* (p 2219) a generalized *staphylococcemia* (p 153) *amebic dysentery* (p 523) or *pleural empyema* (p 2222)

Clinical Manifestations—In the majority of instances the intracerebral brain abscess is *asymptomatic* It may persist silently for a period of many years being discovered only by chance at autopsy or as the result of rupture and the secondary production of a *purulent meningitis* (p 1470)

The *symptomatic intracerebral brain abscess* behaves in the manner of *neoplasm* (p 1419) Its presence is suspected when there arise manifestations of *increased intracranial pressure* (p 1468) often with localizing neurological signs Occasionally the spinal fluid reveals increased pressure a pleocytosis with a predominance of lymphocytes or leukocytes

Treatment—Upon suspicion of the presence of an intracerebral brain abscess the *neurosurgeon* is consulted Persistence of the symptoms of increased pressure demand *exploratory craniotomy* Surface abscesses are clearly visible but deeper collections are revealed only by needling

Drainage of the cavity may be followed by remarkable recovery provided that vital neurological structures are not permanently damaged Prophylactic preoperative and postoperative *anti infective therapy* with sulfonamide (p 88) streptomycin (p 103) or penicillin (p 106) greatly reduce the factors of risk

ABSCESS OF THE SPINAL CORD

Abscesses of the spinal cord are rarely observed They may occur as metastatic manifestations of distant purulent processes or in association with staphylococcemia The clinical manifestations are those of cord neoplasm (p 1430) Differentiation is not important since exploratory laminectomy is required in either instance Preoperative and postoperative therapy with penicillin is conducted in the manner of treatment of brain abscess (p 106)

SUPPURATIVE MENINGITIS

Suppurative infections of the meninges may occur by continuity or through the blood stream

Infection by Continuity—Purulent meningitis may complicate compound fractures of the skull as well as orbital nasal and auditory infections Under any circumstance the patient develops manifestations of meningeal irritation including headache stiffness of the neck a positive Kernig sign and opalescent or frankly purulent spinal fluid The most common invading organisms are staphylococci streptococci and pneumococci

Prophylactic anti infective therapy has greatly reduced the incidence of purulent meningitis Moreover this previously fatal complication now

boasts a highly favorable recovery when prompt and intensive therapy is introduced using sulfonamides and/or penicillin (p 106) The latter may be introduced directly into the subarachnoid space by lumbar puncture Despite the efficacy of anti infective therapy adequate surgical drainage cannot be neglected, more particularly in otogenic and rhinogenic invasions

Hematogenous Purulent Meningitis—Purulent meningitis of hematogenous origin is usually meningococcal pneumococcal or streptococcal Treatment is carried out most successfully in the premeningeal phase employing systemic doses of sulfonamide and penicillin (p 106) Following the secondary meningeal reaction supplementation is essential in the form of intrathecal instillations of penicillin (p 106)

Suppurative Pachymeningitis—Purulent external pachymeningitis may follow local infection of the meninges by extension from an osteomyelitis of the vertebrae or tuberculous caries Anti infective therapy with sulfonamides and penicillin is warranted under any circumstance for control of secondary invading organisms

CHAPTER 74

THE VOLUNTARY NERVOUS SYSTEM DESCRIPTIVE NEUROLOGY

- Peripheral Neuropathies
  - Mononeuropathies
    - Cranial
    - Peripheral
  - Polyneuropathies
- Myelopathies
- Encephalopathies
- Scleroses
  - Subacute Combined Sclerosis
  - Amyotrophic Lateral Sclerosis
  - Primary Lateral Sclerosis
  - Progressive Bulbar Palsy
  - Multiple Sclerosis (Disseminated Sclerosis)
- Syringomyelia
- Syringobulbia
- Paralysis Agitans (Parkinson s Disease)
- Migraine
- Histamine Cephalalgia
- Epilepsy

MANY afflictions of the nervous system are difficult of classification Some are presently recognized as manifestations of *vitamin deficiency* others are obvious *nonbacterial inflammatory processes* the results of *idiopathic degeneration* or *sclerosis* The important conditions of *epilepsy* and *migraine* present no tangible data on which to base nosology

THE PERIPHERAL NEUROPATHIES

Peripheral neuropathies are the commonest neurological disorders They may involve the *cranial* or *vertebral nerves* they may affect one or several nerves

The *mononeuropathies* whether peripheral or cranial include neuralgias causalgias and paralytic syndromes They are most often the products of local pathology but may be of systemic origin *Polyneuropathies* result from exogenous chemicals or drugs but they may follow disturbances of endogenous metabolism

ANATOMY

The Cranial Nerves.—The cranial nerves are twelve in number The *first second* and *eighth* cranial nerves have purely sensory functions and are concerned respectively with smell vision and hearing the *third fourth* and *sixth* regulate extra-ocular movements the *eleventh* controls the motor functions of the neck and shoulder and the *twelfth* activates the tongu

The *vagus* or *tenth* cranial nerve is the link with the cholinergic portion of the involuntary nervous system The *adrenergic subdivision* has no corresponding intracranial representation

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The *fifth seventh* and *ninth cranial nerves* are mixed in their functions The *trigeminus* is concerned with mastication and facial sensation the *seventh* nerve innervates the facial muscles and shares with the *ninth nerve* the sensations of taste the *glossopharyngeus* has motor functions concerned with the musculature of the pharynx

The Spinal Nerves —The spinal nerves are paired to supply symmetrical portions of the body and extremities They number eight in the *cervical* twelve in the *thoracic* five in the *lumbar* five in the *sacral* and one in the *coccygeal* regions The lower cervical and upper thoracic branches join to form the *brachial plexus* which is concerned with the innervation of the upp r extremity the lower lumbar and the upper sacral cord unite in the *lumbosacral plexus* for the legs

Each spinal nerve splits into an *anterior* and *posterior root* as it enters the vertebral column The *anterior root* connects with the *motor horn cell pool* which is situated in the anterior gray matter the *posterior root* connects with the *sensory horn cell pool* in the

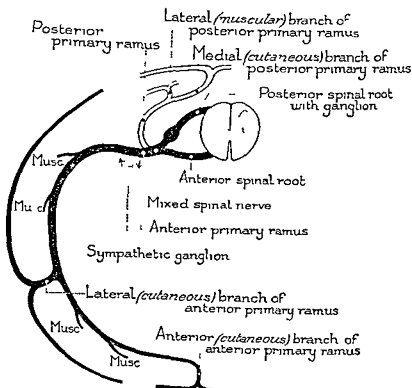


Fig 277—The components of a segmental nerve For purposes of clarity the posterior primary ramus is indicated in outline *Musc* refers to the muscular branches given off by the anterior primary ramus *

posterior gray matter Anterior and posterior horn cell pools are connected in turn each with the other as well as with their fellows of the opposite side and with similar horn cell pools of other segments This arrangement provides for symmetry and coordination of function.

PHYSIOLOGY

The final common pathway is the basic physiological unit of the nervous system *Sensory receptors* are situated in the skin, bones joints and muscles They register touch pain, temperature vibration deep pressure and the sense of position in space Their stimuli are carried through peripheral nerves and into posterior horn cell groups They initiate responses which originate in the anterior horn cell group and pass out through the anterior root and the peripheral nerves to effect adequate responses usually muscular Any lesion of the final

* Haymaker and Woodhall Peripheral Nerve Injuries

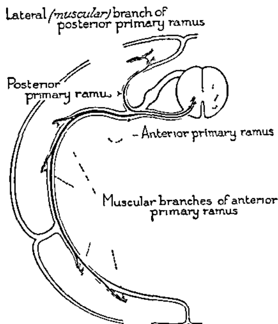


Fig 278—A segmental nerve illustrating the routes traveled by efferent fibers to muscles

Medial (cutaneous) branch of posterior primary ramus
Lateral branch Medial branch
Posterior branch
Po t r o column
Lateral (cutaneous) b anch of ant r or pr mary ramus
Ant p nothalam c t a t
Lat sp nothalamic tract
Anterior branch
Anterior (cutaneous) branch of anterior primary ramus
Lateral Medial

Fig 278A—A segmental nerve showing the course of sensory neurons from the periphery to the spinal cord The fibers ind cated by the broken l ne convey deep sensibil ty thos in solid black touch and those shown as dotted lines pain and thermal sensib lity

Haymaker and Woodhall Peripheral Nerve Injuries

common pathway destroys the sensory motor and reflex activities of the individual peripheral nerve and blocks stimuli which might traverse the cord or initiate in the higher centers

The Motor Pathway —The motor activities of the "final common pathway are influenced by *voluntary involuntary* and *postural elements* which are transmitted by separate pathways through the spinal cord Voluntary movement is conducted along the *pyramidal tracts* whose interruption produces spasticity hyperreflexia and abnormal reflexes such as the Babinski Modifications occur through the *extrapyramidal* and the *spinocerebellar elements* Interruptions of the former result in *ataxia* and *abnormal movements* disturbances of the *cerebellar pathway* are marked by *hypotonus* and further *ataxia* as summarized in Table 96

The Sensory Pathways —The important sensory pathways are the *spinothalamic tracts* and *posterior columns* The former is primarily concerned with the recording of *pain* and *temperature* whereas the latter deals essentially with the sensations of *touch vibration* and *position Sensory dissociation* results from the prompt crossing of the spinothalamic tract across the cord whereas the decussation of the fibers of the posterior columns does not occur until the length of the cord has been traversed Thus involvement of *both pathways* may produce a *contralateral* loss of the sensations of pain and temperature but an *ipsilateral* anesthesia ataxia and pallanesthesia as illustrated in Table 96

PAIN

The healthy, normal human being registers few sensory intimations of the existence and the manifold activities of his carcass In the course of the average day, hunger and thirst are experienced the individual becomes fatigued and then rested he is required to urinate and defecate a sexual urge develops which may or may not be gratified Aside from these registrations there are certain sensory messages that bespeak difficulty Of these the most important is pain

THE FUNCTION OF PAIN

Pain is said to be a pleasurable sensation to certain distorted and perverted individuals who enjoy inflicting pain (sadists) or recording pain (masochists) The remainder of the relatively normal human population regards pain as an unpleasurable sensation to say the least Those of a religious turn of mind interpret pain as a punishment or expiation for sin

The practitioner recognizes pain as a fundamentally protective mechanism The child who experiences the pain of a burn keeps away from the fire the adult with abdominal cramps has no interest in food the patient with angina pectoris discontinues activity and rests until the pain has passed In these examples pain has served to prevent further trauma and to secure rest for an anguished part or organ

Pain serves to localize clinical abnormalities Though the sensation may at times be misleading the sufferer reports with subjective accuracy that it is the ear leg or tooth that aches or is sensitive The diagnostician is directed to a more careful investigation of the painful part

Pain may indicate a therapeutic principle The hunger pain of gastroduodenal ulcer emphasizes the principle of frequent feeding Each of us has learned by experience that certain skeletal and muscular discomforts are aggravated by use while others are ameliorated by active motion

Direct Pain —Direct pain is recorded by stimulation of specific nerve endings in skin subcutaneous tissues fascial coverings of muscles and tendons synovia surrounding joints periosteum and parietal lining surfaces of skull and vertebrae and thoracic abdominal cavities

Tegumentary and mucous surfaces are the most richly provided with sensory nerve endings easily demonstrable by pinprick The sensitivity of other structures is best noted in surgical procedures under local analgesia The surgeon may handle muscle tendon cartilage and bone without registration of pain He produces painful stimuli by manipulation of fascial coverings synovia or periosteum Within the body cavities there is sensitivity of parietal membranes but insensitivity of visceral coverings and the viscera themselves The practitioner who performs needling procedures (thoracentesis or abdominal paracentesis) observes that pain occurs when the needle is pushed through skin and again at parietal pleura or peritoneum

The Pathway for Direct Pain—The pathway for direct pain is initiated at punctiform nerve endings throughout the sensitive tissue *Afferent nerve fibers* which transmit sensation from nerve endings gather to form *posterior roots* which terminate in the grey matter of the cord in the region of the posterior column

From the posterior column there is an axone relay which crosses to the opposite side in the grey commissure to enter the lateral funiculi and form the *lateral spinothalamic tract* The painful sensation is carried by the spinothalamic tract to a center in the *medulla* whence it is relayed to the *thalamus* finally registering in the *cerebral cortex*

The Nerve Endings of Painful Sensation—The specific nerve endings which transmit the impulse of pain have a punctiform distribution They vary in their numbers and sensitivities in different regions of the body The finger tips for example are exceedingly attuned to the slightest stimulus

Pain sense is *dissociated* from other peripheral sensations as illustrated in a cocainized area where the practitioner may satisfy himself that the sense of touch is retained though the pain sense has been greatly diminished or even abolished The term *local anesthesia* thus is a misnomer since actually the cocainized area is under *local analgesia*

The Spinal Pathway for Pain—The spinal pathway for pain is so devised that it dissociates pain from touch and produces crossed sensory disturbances

Dissociation of sensation in the cord is best seen in syringomyelia (p 1505) whose lesions involve chiefly the central grey and contiguous portion of the white matter As a result the patient with syringomyelia may present loss of pain and temperature sense in an area which retains the ability to recognize touch Such patients suffer from burns because of inability to recognize that a steam pipe or a hot water bottle which gives a tactile sensation is sufficiently hot to cause a superficial or even a deeper burn

Crossing of the pain sense in the cord is demonstrated in unilateral cervical lesions in which loss of pain sensation occurs on the side opposite to the lesion

The Medullary Pain Center—Nerve endings of the spinothalamic tract probably terminate in a pain center in the medulla The existence of this pain center is clinically suggested by the daily experiences with morphine and its derivatives Without producing local analgesia or unconsciousness the opiate gives blessed relief to all types of pain The site of action

TABLE 96—REACTION PATTERNS OF INJURIES

Level	Muscle Tone	Muscle Strength	Muscle Volume	Abnormal Movements	Superficial Reflex	Deep Reflexes
Primary muscle dystrophy	Diminished	Diminished	Diminished or increased	None	Diminished	Diminished
Peripheral nerve	Diminished	Diminished	Diminished	None	Absent	Absent
Posterior root or horn	Diminished	Diminished	Diminished	None	Absent	Absent
Anterior root or horn	Diminished	Diminished	Diminished	None	Absent	Absent
Pyramidal tract (corticospinal)	Increased	Diminished	Normal	None	Diminished	Increased
Extrapyramidal tract	Increased	Diminished	Normal	Greatly increased See Note 2	Normal	Normal
Cerebellar tract	Diminished	Diminished	Normal	Increased	Normal	Diminished
Spinothalamic atrophy	Normal	Normal	Normal	None	Normal	Diminished
Spinothalamic irritation	Normal	Normal	Normal	None	Normal	Normal
Posterior column atrophy	Normal	Normal	Normal	None	Normal	Normal
Posterior column irritation	Normal	Normal	Normal	None	Normal	Normal

NOTES ON TABLE 96

1 The abnormal reflexes of the pyramidal tract origin include the Babinski and its modifications and clonus

2 The abnormal movements of extrapyramidal origin include tics tremors chorea and athetoses

3 Cerebellar ataxia is equally severe whether the eyes are open or closed

4 Posterior column ataxia is increased by closing the eyes

5 The paresthesias of posterior column irritation include pains such as the lightning pains of tabes and abnormal sensations such as tingling burning itching

Abnormalities of *touch*—Hypesthesia hyperesthesia anesthesia and paresthesia

Abnormalities of *pain*—Hypalgesia hyperalgesia and analgesia

Abnormalities of *temperature*—Thermhyperesthesia and thermanesthesia

Vibration—Pallanesthesia

Dissociation—Loss of the sensations of pain and temperature but maintenance of tactile sensations

is undoubtedly medullary where a center for pain may well be postulated

Thalamic Center—Clinical experience also suggests that there is a pain center in the thalamus Lesions in this area produce the so called thalamic syndrome (Dejerine Roussy) characterized by excruciating

TO PERIPHERAL NERVES CORD AND TRACTS

Abnormal Reflexes	Touch	Pain	Temperature	Vibration	Position	Ataxia	Distribution
Absent	Normal	Normal	Normal	Normal	Normal	None	Irregular
None	Absent	Absent	Absent	Absent	Absent	None	Neuritic
None	Absent	Absent	Absent	Absent	Absent	Present	Segmental
None	Normal	Normal	Normal	Normal	Normal	None	Segmental
Present See Note 1	Normal	Normal	Normal	Normal	Normal	Present	Ipsilateral below medulla
None	Normal	Normal	Normal	Normal	Normal	Present	
None	Normal	Normal	Normal	Normal	Normal	Present See Note 3	Ipsilateral
None	Diminished	Absent	Absent	Normal	Normal	None	Contralateral
None	Diminished	Increased	Increased	Normal	Normal	None	Contralateral
Normal	Diminished	Normal	Normal	Absent	Absent	Present	Ipsilateral
Normal	Paresthesia See Note 6	Normal	Normal	Absent	Absent	See Note 4 None	Ipsilateral

paroxysmal pain in an area that is apparently normal so far as local pathology is concerned Thalamic pain is not allayed by the local use of cocaine nor the medullary action of morphine suggesting a supramedullary center for pain serving as a final relay station before registration in the cerebral cortex

Referred Pain—The surgeon who employs local anesthesia readily demonstrates the absence of *direct pain in muscle tendon cartilage bone* visceral lining membranes and viscera in cranial thoracic and abdominal cavities Despite the absence of direct pain sensation disturbances of these structures and areas are capable of producing pain by a referred mechanism

Visceromotor and Viscerosensory Reflexes—Referred pain may be produced by spasm of smooth muscle as illustrated by intestinal cramps or labor pains (visceromotor reflex) It may also arise from inflammation of the visceral surface (appendicitis pleuritis meningitis) through the mechanism of a viscerosensory reflex

The viscerosensory reflex registers subjectively and gives objective signs through spasm of striated muscle of the specific dermatome (right rectus spasm in acute appendicitis)

Accurate and Misleading Projection of Referred Pain—The projection of visceral pain may be accurate, as in the localization of the sensation in the right lower quadrant in appendicitis or in the precordium with cardiac difficulties It may on the other hand be bizarre inaccurate and misleading, as for example the pain in the right shoulder with gallbladder disease *in the left arm with angina pectoris in the testicles with renal stones* and in the knee with disturbances of the hip joint

The Pathway and Mechanism of Visceral Pain—The explanation of the accurate and misleading projection of referred pain rests in an understanding of the pathway and mechanism of referred pain

Each spinal segment supplies a well delineated area of the skin (dermatome) with sensory and motor fibers These same segments also supply autonomic nerve fibers to the various viscera the distribution of which is indicated in Table 97

The stimulus that arises in a diseased organ enters the cord through autonomic fibers and produces an irritability of the cells that also receive direct impulses from the corresponding dermatome Excitation of the spinal cells evokes stimuli that travel in two directions Central stimuli pass to the *thalamus and cerebral cortex there to register as pain* referred to the dermatome whose spinal nerves correspond to the autonomic nerves of the inflamed organ (viscerosensory reflex) Meantime in the local spinal segment the motor efferents are stimulated giving rise to the rigidity of the locally innervated striated muscle Excitation of the sensory afferents produces areas of segmental hyperalgesia (Head zones)

The inflamed viscus through the referred mechanism has produced pain hyperalgesia tenderness and rigidity in the corresponding dermatome which may or may not be in direct anatomical relationship

Referred pain may also arise in an anatomically normal viscus as for example the colic that accompanies disorders of the hollow viscera (stomach intestines urinary passages gallbladder uterus or tubes) The stimulus to pain in an anatomically normal viscus arises when the wall is stretched in the region of a nerve terminal This is best illustrated by considering the physiological disturbances of the stomach When the stomach is empty strong peristaltic contractions arise which give origin to the sensation of hunger These contractions may be greatly increased producing nausea and vomiting but there is no pain so long as there is no distention of the gastric wall Should the pylorus be obstructed the resulting distention of the stomach produces stretching of the wall and referred pain So far as is known the visceromotor reflex manifested by spasm and segmental hyperalgesia (Head zones) are rarely if ever produced by colic alone Their presence strongly suggests anatomical disease (particularly inflammation) Thus spasm of a right rectus indicates a possible appendicitis rather than a bellyache

Recapitulation—1—Knowledge of the mechanism and pathways of direct and referred pain is important to the practitioner in the diagnostic approach and therapeutic management of painful conditions

2—From the diagnostic standpoint the subjective complaint may be caused by direct or distant referred mechanisms Thus left shoulder pain may be due to a subdeltoid bursitis but it may be re-

ferred from a diaphragmatic pleurisy or from a cardiac source Right shoulder pain may be due to a localized myositis or arthritis but it may also be caused by gallbladder colic Pain in the testicle may be due to orchitis but it may also represent renal stone Precordial pain may be derived from a fractured rib or referred from coronary occlusion

3 —Knowledge of the pathway of pain assists in treatment Pain may be allayed locally by heat or cold massage counter irritation immobilization or pressure Local analgesia may be temporarily obtained by cocainization and more lastingly produced by alcohol injection of a nerve ganglion the posterior root or plexus It may be permanently relieved by section of the root

TABLE 97—THE DISTRIBUTION OF AUTONOMIC NERVE FIBERS FROM SPINAL SEGMENTS TO VISCERA*

Viscus	*Spinal Segments*
Lungs	1–7 dorsal mostly 2–5 dorsal
Heart	3–5 cervical 1–8 dorsal predominantly on left side sometimes bilateral
Esophagus	Mainly 5 dorsal also 6 7 and 8 dorsal
Breast	4 and 5 dorsal
Stomach	7 8 and 9 dorsal usually bilateral
Intestine	9–12 dorsal, bilateral or on left side only
Liver	8–10 dorsal on right side
Gallbladder	Mostly 8 and 9 dorsal also 5–7
Kidney	Mostly 10 dorsal, also 11 and 12 dorsal and 1 lumbar
Ureter	11 and 12 dorsal and 1 lumbar
Testis	10 dorsal
Epididymis	11 and 12 dorsal
Bladder	11 and 12 dorsal and 1 lumbar also 3 and 4 sacral
Prostate	10 and 11 dorsal also 1–3 and 5 sacral
Ovary	10 dorsal
Fallopian Tubes	11 and 12 dorsal
Uterine Cervix	11 and 12 dorsal and 1–4 sacral
Uterine Body	10 dorsal to 1 lumbar

After Head from Brain W R Diseases of the Nervous System ed 2 New York Oxford University Press

4 —Relief of pain also may be accomplished centrally by the action of the specific drugs such as the analgesics antipyretics demerol or the narcotics of the opium family Finally in desperate situations it may be necessary to produce general unconsciousness by the use of anesthetics in order to relieve a tortured individual of intractable pain

5 —The therapeutist aims to effect maximum analgesia with minimum side effects He uses first the simpler local measures Not until he is driven to it does he employ drugs that act centrally particularly narcotics which may depress the respiratory center and produce addiction If pain can be strictly localized surgical procedures such as injections with alcohol and posterior root section may be a tremendous boon

6 —In the management of painful conditions the immediate interest of the practical clinician is relief of the local disturbance The general considerations that have been outlined above serve as the

Accurate and Misleading Projection of Referred Pain—The projection of visceral pain may be accurate as in the localization of the sensation in the right lower quadrant in appendicitis or in the precordium with cardiac difficulties It may on the other hand be bizarre, inaccurate and misleading as for example the pain in the right shoulder with gallbladder disease in the left arm with *angina pectoris* in the testicles with renal stones and in the knee with disturbances of the hip joint

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TRIGEMINAL NEURITIS

Irritative phenomena involving the fifth cranial nerve may result in *trigeminal neuralgia sphenopalatine neuralgia* or *trismus* Loss of function may be followed by *anesthesia* of the skin of the face the anterior half of

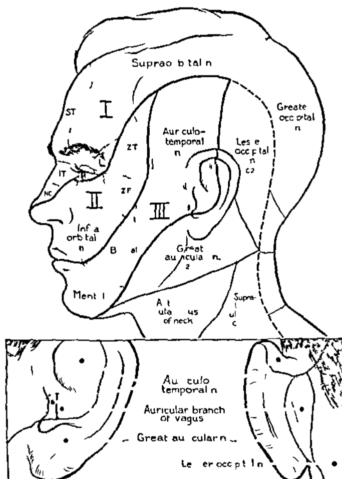


Fig 279 —Diagram of the cutaneous fields of the head and upper part of the neck The three divisions of the trigeminal nerve (I ophthalmic II maxillary III mandibular) are indicated by heavy lines and their respective subdivisions by light broken lines The conjunctivae are innervated by the ophthalmic division Abbreviations refer to the following nerves B buccal IT infratrochlear L lacrymal NC external nasal branch of the nasociliary ST supratrochlear ZF zygomaticofacial ZT zygomaticotemporal The lateral and superior boundaries of the posterior primary rami are indicated by broken lines The inset shows trigeminal and vagal cutaneous fields of the region of the external ear and the external auditory meatus

the scalp the mucous membranes of the lips mouth tongue pharynx accessory nasal cavities teeth and portions of the dura mater the cornea and conjunctiva Abolishment of motor function is associated with *paralysis* of the muscles of mastication and may be central or peripheral

* Haymaker and Woodhall Peripheral Nerve Injuries

enunciation of fundamental principles For purposes of specific information relative to differential diagnosis and treatment of the various conditions management is discussed in this text under the classification of topographic localization (*pain in the head, in the ear in the eye in the abdomen in the chest in the testicles etc*) or physiological function (*dysuria dysphagia dyspepsia etc*)

THE MONONEUROPATHIES

The mononeuropathies are 'reaction patterns that result from pathological involvement of any single nerve whether cranial or peripheral Irritations produce the *neuralgias* or *causalgias* dissolution of continuity causes *atrophy* or *paralysis*

OLFACTORY NEURITIS

Neuritis of the olfactory nerve (I) is most often peripheral in origin Central irritations due to a neoplasm in the region of the uncus or to cerebrovascular disease may produce hallucinations of smell or *parosmia* The latter may also be functional as part of a major *hysteria* (p 1353)

Peripheral involvements result in *hyposmia* or *anosmia* (p 2190) Common examples are impairments or destructions of the nerve endings due to *upper respiratory infection* chronic *nasal sinusitis* with *polyp* formation *atrophic rhinitis vasomotor rhinitis* of allergic origin excessive use of *tobacco* sniffing of *cocaine* and *occupational exposure* to heavy fumes as in the handling of wine Infrequently the olfactory nerve is damaged as the result of a *fracture* through the cribriform fossa meningeal thickening a tumor of the frontal lobe a meningioma or a fibroma involving the nerve or its sheath The majority of peripheral afflictions produce a unilateral loss of sensation which is only manifest when tests are directly made pertinent to this finding

Diagnosis and Treatment—The patient with a unilateral anosmia warrants *specialist consultation* for investigation of a remediable lesion of the nerve itself or of the structures of the anterior fossa in the near vicinity *Neurosurgical consultation* is also warranted when there are hallucinations of smell A bilateral and complete anosmia calls for the opinion of the *rhinologist* since the affliction is almost invariably related to a mucous membrane disturbance

OPTIC NEURITIS

See p 1640

OCULOMOTOR NEURITIS

See p 1645

TROCHLEAR NEURITIS

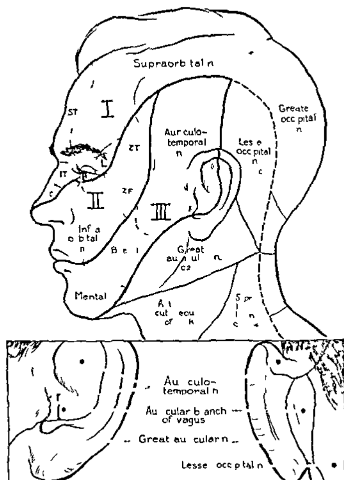
See p 1647

ABDUCENS NEURITIS

See p 1647

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TRIGEMINAL NEURALGIA (TRIFACIAL NEURALGIA TIC DOULOUREUX)

Trigeminal neuralgia occurs as a symptomatic or idiopathic complaint In the former instance the disturbance is the result of a neoplasm inflammation or gliosis of the stem, cerebellum or cerebellopontine angle (p 1496) More often the affliction is not associated with demonstrable pathological disease and represents an *irritation of the gasserian ganglion* its roots and its connections with other nuclear cells

Clinical Manifestations—The clinical manifestations of trigeminal neuralgia are characterized by the periodic appearance of transitory attacks of *excruciating pain* involving the sensory areas supplied by one two or three of the branches of the trigeminus In the idiopathic form the condition is seen most often in middle life The pains are of indescribable violence and the sufferer winces and contorts his face in agony Each attack is of short duration but a repetition of the anguish may occur in a few seconds a few minutes or a few hours There may be periods of intermittence of variable length

The attacks of pain are brought on by apparently trivial sensory phenomena such as eating drinking talking exposure to cold washing the face shaving attempts to rest the head on a pillow a loud sound or light touch to trigger zones' which are most often found around the nose and mouth The areas of the *mandibular* and *superior maxillary branches* are affected most commonly but the *ophthalmic division* may also be involved

An idiopathic trigeminal neuralgia is not accompanied by objective neurologic findings Efforts should be made to demonstrate the presence of sensitizing phenomena such as *dental infection* or *inflammation of the nasal accessory sinuses* The presence of *increased intracranial pressure* (p 1491) *tinnitus deafness ataxia* or *areas of anesthesia* requires the practitioner to abandon the diagnosis of an idiopathic neuralgia and refer the patient to the specialist consultant for investigation of an intracranial lesion

Treatment—Idiopathic trigeminal neuralgia despite its tendency to be intermittent does not permanently disappear except as the result of therapy by injection or surgical intervention *Inhalations of trichlorethylene* (p 3864) are occasionally helpful in mild manifestations Palliative measures include *peripheral avulsion* and *injections of alcohol* into the branches of the nerve as they leave the skull The efficacy of avulsion is short lived and the procedure cannot be repeated Deep injections of alcohol which may be repeated prove successful in approximately 75 per cent of patients

The radical treatment consists of *injections of alcohol* into the *gasserian ganglion* or of *subtotal to complete section of the sensory root* through a transtemporal or suboccipital approach In expert hands surgical procedures have minimum mortality and maximum promise of success

SPHENOPALATINE NEURALGIA (SLUDER)

Sphenopalatine or vidian nerve neuralgia bears many resemblances to tic douloureux It is differentiated in that it produces a lower half headache with boring and burning pain in the superior maxillary region and radiation down into the neck and shoulder It may be associated with inflammatory disease of the nasal accessory sinuses and sometimes accom

panies an *ophthalmic migraine* (p 1506) or impaction and infection of maxillary teeth

The *diagnosis* of Sluder s neuralgia is substantiated by the comfort that is afforded by cocainization of the sphenopalatine ganglion Permanent relief may follow the injection of alcohol and phenol by the experienced expert

SENSORY PARALYSIS OF THE TRIGEMINUS

Sensory paralysis of the trigeminal nerve results in distributional hypesthesia or anesthesia The site of the lesion may be peripheral or central The former may result from affections of the branches of the nerve as the result of inflammatory processes in the deep tissues of the face or mouth inflammations or neoplasms involving the cranium or the bone that surrounds the various foramina localized meningitis or neoplasms of the pons cerebellum acoustic nerve or the nerve sheaths (von Recklinghausen s disease) Penetrating wounds and fractures of the base of the skull may lacerate the nerve the ophthalmic division is exposed to injury by aneurysms of the internal carotid artery cavernous sinusitis and neoplasms of the hypophysis

Central disturbances dependent upon intra axial involvement include vascular lesions neoplasms of the medulla or pons inflammations such as syringobulbia and epidemic encephalitis and scleroses especially of the disseminated variety

Clinical Manifestations —Sensory paralysis of the trigeminus is characterized by reduction or loss of the sensations of touch pain and temperature in the areas supplied by the nerve (p 1481) Trophic changes may result in disturbances of the senses of *smell* and *taste* despite the fact that the former is regulated by the first cranial nerve and the latter by the seventh and ninth In a zoster the characteristic vesicular eruption (p 435) is observed along the path of the involved root Ophthalmic afflictions whether due to herpes or operative procedures have extreme gravity since they may be associated with ulcerations of the cornea resulting in permanent opacity and disturbances of vision (p 1626)

Peripheral afflictions are marked by the most complete sensory manifestations dissociation suggests a pathologic process within the neuraxis and sensory disturbances limited to alteration in tactile discrimination point to a more central lesion

Treatment —The management of anesthesia of the trigeminus is directed towards the elucidation of the cause and its possible elimination Disturbances involving the ophthalmic division call for prophylactic therapy directed at the cornea (p 1628) Consultation with the ophthalmologist is warranted and it may be necessary to suture the upper lids so that the cornea is protected against ulceration

MOTOR TRIGEMINAL IRRITATION

Irritation of the motor branch of the trigeminal nerve results in the production of *trismus* This phenomenon is seen most often in *chilling* when it has little significance On the other hand it is of ominous import as an indication of *tetanus* (p 294) *rabies* (p 439) and *strychnine poisoning* (p 3869)

Tremors of the *muscles of mastication* occur in *paralysis agitans* (p 1505) and *epidemic encephalitis* (p 441)

MOTOR TRIGEMINAL PARALYSIS

Motor paralyses of the muscles of mastication occur peripherally or centrally In the former instance the disturbance is unilateral and may not seriously interfere with the function of chewing Bilateral central paralysis occurs infrequently as a manifestation of *encephalitis* (p 441) or a *supranuclear neoplasm*

PROGRESSIVE FACIAL HEMIATROPHY

Progressive facial hemiatrophy probably results from involvement of the fibers of the involuntary nervous system that accompany the divisions of the trigeminal nerve The condition is apparent from a progressive atrophy of the bone cartilages and soft tissues of the face despite the fact that the muscles show no evidences of degeneration The condition is progressive and may follow injuries to the head and face local infections exposure to cold erysipelas and osteitis of the jaw *Treatment* is of little avail

BELL S PALSY

A peripheral paralysis of the seventh cranial nerve may be *symptomatic* but it is more often *idiopathic* Demonstrable *organic* causes include inflammations and suppurations that involve the nerve at the base of the skull tumors of the acoustic nerve, endotheliomas of the meninges, fractures of the base of the skull and operative procedures involving the mastoid cells In the fallopian canal the nerve may be damaged by otitis media and osteomyelitis of the petrous portion of the temporal bone These major disturbances produce clinical phenomena other than the facial paralysis those which involve the organs of hearing are associated with derangement in the acoustic functions

Much more frequently the Bell s palsy results from some inconsequential circumstance such as the application of ice to the side of the neck or face exposure to draughts or an electric fan or following a hair wash The mechanism of the paralysis is far from clear but it is thought that a perilymphangitis in the unyielding bony canal produces a pressure neuritis

Clinical Manifestations—The idiopathic Bell s palsy comes on rapidly in the course of a few hours It may be preceded by *pain* in the area of the distribution of the nerve but the distinguishing characteristic is *distortion of the face* and *inability to whistle* or *close the eye* The mouth is drawn toward the normal side where its corner is elevated The wrinkles of the forehead and the nasolabial folds flatten The eye appears more widely open on the involved side and the lower lid is often slightly everted so that the mucous membrane is visible Voluntary movements such as the attempts to show the teeth wrinkle the forehead or whistle greatly increase the asymmetrical appearance Saliva drools from the paralyzed side and tearing is noted from the involved eye

In zoster the characteristic vesicles may be apparent on the auricle or within the external auditory canal Idiopathic Bell s palsy is not associated with tinnitus or disturbances in hearing and equilibration

Treatment—The treatment of idiopathic Bell s palsy is most unsatisfactory The patient is assured that experience has shown that *spontaneous recovery* is likely to occur The eye is protected with a patch and frequent washings with saline solution The administration of *salicylates* (p 3834) or *iodides* (p 612) is conventionally urged on a purely empiric basis In our experience drugs are of little if any efficacy Local counterirritation can only add to the difficulties but the application of warm wet dressings provides the patient with something to do Doses of *strychnine* (p 3868) are ordinarily prescribed but they accomplish nothing *Electrical treatment* with galvanism or faradism may be suggested in order to keep up muscle tone diathermy (p 3788) is highly regarded by enthusiasts for this form of therapy

SUPRANUCLEAR PARALYSIS OF THE FACIAL NERVES

Supranuclear paralysis of the facial nerve is fortunately uncommon It is differentiated from peripheral involvement as seen in Bell s palsy by the failure to involve the superior facial branches which supply the orbicularis and the muscles which produce wrinkling of the forehead

Supranuclear paralysis of the facial nerve is usually associated with an *ipsilateral hemiplegia* (p 1427) and may be due to tumors of the pons hemorrhage into the internal capsule or an encephalitis Associated paralysis of the abducens places the lesion in close proximity to the pons

Symptomatic Bell s palsy is treated by an attack on the more fundamental condition Operative anastomoses have been attempted by expert neurosurgeons without consistent success by any of several methods

NEURITIS OF THE INTERMEDIUS (WRISBERG)

The afferent fibers of the intermedius nerve of Wrisberg convey sensations of taste through the glossopharyngeal nuclei efferents join the submaxillary ganglia and transmit secretory impulses to the submaxillary and sublingual glands With inflammations of the *geniculate ganglion* most often seen in *herpes zoster* (p 435) vesicles appear in the external auditory canal and may implicate the motor fibers of the facial nerves Taste is lost over the anterior portion of the tongue Associated involvement of the *auditory nerve* produces tinnitus vertigo diminution in hearing nausea and vomiting

COCHLEAR NEURITIS

Paralysis of the cochlear portion of the auditory nerve (VIII) is almost invariably of peripheral origin The definitive *organic causes* include tumors of the nerve trunk or the cerebellopontine angle (p 1426) a toxic *neuritis* occurs in cinchonism (p 862) and salicylism (p 3834) *inflammations* occur in meningitis scarlet fever typhoid fever tabes dorsalis multiple sclerosis malaria mumps and influenza *Hemorrhages* are encountered in the bleeding diatheses (p 1108) and the nerve termination is often affected in *labyrinthine disease* and *otitis*

Clinical Manifestations—Paralysis of the cochlear portion of the acoustic nerve results in *impairment of hearing* This type of deafness is to be distinguished from that which results from afflictions of the middle ear *Nerve deafness* is characterized by lessened bone conduction but relatively greater air conduction when tested with the tuning fork With the

Rinne test the vibrating tuning fork is heard when held opposite the external auditory meatus after it is no longer recognized when placed upon the mastoid process In the *Weber test* with the vibrating tuning fork placed on the vertex of the skull and occlusion of one and then the other ear the sound is best heard on the normal side (*negative Weber*) In middle or external ear involvement lateralization of sound is on the side of the impaired hearing

Treatment—The treatment of cochlear nerve paralysis involves the elimination of possible etiologic factors such as quinine and the salicylates Otherwise management is directed at the more fundamental disturbance

VESTIBULAR NEURITIS (MENIERE S DISEASE ACUTE LABYRINTHITIS)

Meniere s disease is an acute disturbance of the labyrinth As with other forms of irritative neuritis, the cause is most often *idiopathic* rather than demonstrably organic In the latter instance it may be the result of inflammation of or hemorrhage in the labyrinth as the result of the various infectious diseases especially syphilis and the hemorrhagic phenomena More often Meniere s disease like trigeminal neuralgia occurs recurrently and without obvious pathological manifestations

Clinical Manifestations.—The acute attack of Meniere s disease is characterized by the sudden onset of intense dizziness vomiting disturbances in equilibrium and nystagmus Except for the labyrinthine phenomena the examination reveals no significant abnormality and the attack passes only to recur after a variable interval In our experience many of the patients with Meniere s disease suffer psychogenic disturbances

Treatment—The patient with Meniere s disease may be assured that recovery will be noted within a few hours to a few days with or without treatment Bed rest is required in a quiet and darkened room sedatives and hypnotics are administered in generous doses

Intranasal Treatment—If there is a history of a recent respiratory infection or the patient is known to have chronic inflammation in the nasal or auditory passages specialist consultation is advisable A blockage of the *eustachian tube* may be alleviated by use of vasoconstrictor substances introduced intranasally (p 2027) or actual catheterization of the passageway by an experienced expert

Dietotherapy—Those who incline to the theory of the metabolic origin of Meniere's disease have demonstrated a *retention of sodium* in the body Following this clue they suggest the use of a restricted sodium diet of low salt content This list of foods to be avoided or taken sparingly includes salt meat and fish salted bread and crackers and salt butter Carrots clams condensed milk raisins caviar peas olives spinach cheese endive oysters lima beans beets buttermilk cantaloupe cauliflower celery chard dried cocoanut dried currants dates figs horseradish kohlrabi limes musk melons peanuts peach mustard pumpkin radishes rutabagas strawberries turnips turnip tops and escarole are on the restricted list All foods are prepared and served without salt

The dietary routine is supplemented by the administration of 3 gm *ammonium chloride* taken three times daily for three days with a two day holiday

Potassium Therapy—The success of the low sodium routine suggested

that the favorable outcome was attributable to a relative *hyperpotassemia* As a substitute for the cumbersome diet 1 gm potassium chloride has been administered at two hour intervals without dietary restrictions A teaspoonful of *25 per cent potassium chloride solution* represents an approximate dose and a trial for three weeks is recommended

Surgery—Meniere s disease can be relieved by partial root section of the involved auditory route The procedure is performed through a cerebellar approach and is reserved for incapacitating examples which fail to respond to low sodium or high potassium therapy

Miscellany—As in the case of all self limited diseases many cures for Meniere s disease are reported They include injections of histamine thiamine chloride niacin strychnine and neurophosphates

SEASICKNESS

The syndrome of seasickness is included with the present material since there is a suggestion that the disturbance results from overstimulation of the equilibratory mechanism of the internal ear The clinical manifestations are all too familiar and consist of epigastric discomfort anorexia salivation headache dizziness weakness a greenish pallor dejection and bouts of nausea and vomiting

Treatment—Despite the many adherents to the *psychogenic theory* of the origin of seasickness we incline to the belief that the disturbance is functional in a labyrinthine sense

Prevention—Prophylaxis by the generous use of *sedatives* and *hypnotics* prior to the voyage is helpful More recently repeated doses of *amphetamine sulfate* (benzedrine) have won adherents though the resultant sleeplessness and restlessness add to the awareness of discomfort Many experienced travelers favor inhalations of 100 per cent oxygen or the use of hyoscine (gr 1/100) with or without *barbiturates* or *chlorbutanol*

A time honored prescription consists of *chloral* and *sodium bromide* as undernoted

℞	Sodium Bromide	15 0
	Chloral Hydrate	8 0
	Syrup of Oran e Peel q.s a d	60 0

Sig 1 teaspoonful every 3 hrs for 4 doses preceding the voya e

Seasickness may be mitigated by a *fast* of at least twelve hours and a *purge* taken at the beginning of the fast On boarding ship the susceptible patient is ordered to *recline in bed* for at least the first twenty four hours of the trip The doses of the *bromide and chloral mixture* are continued food intake is limited to tea and toast ingested in a *semirecumbent position* If the diet of tea and toast becomes monotonous carbonated drinks such as dry ginger ale or champagne act as carminatives and combat dehydration The room is freely *ventilated* and *darkened* The *ears* are plugged with cotton and a tight *abdominal binder* is applied particularly in the visceroptotic (p 3488)

Active Treatment—With the onset of seasickness the prophylactic measures are supplemented by intravenous injections of 50 cc of 50 per cent *dextrose* and subcutaneous hypodermics of 1/80 grain of *atropine sulfate* or 1/100 grain of *scopolamine hydrobromide* (*hyoscine*) The addition

of *strychnine sulfate* despite its advocates seems irrational *Amphetamine sulfate* (*benzedrine*) is used in larger dosages, such as 10 to 20 mg with reported spectacular improvement in 80 per cent of those treated Inhalations of 100 per cent *oxygen* are recommended and other favored measures include the generous use of *chlorbutanol* (chloretone) in doses of 6 to 9 grains Injections of morphine seem to be hazardous because of the frequent tendency of morphine to produce vomiting as an untoward effect

GLOSSOPHARYNGEAL NEURALGIA

The *petrosal and jugular ganglia* of the ninth nerve may be affected in the manner of a trigeminal neuralgia (p 1482) The disturbance is most often idiopathic and rarely, if ever, associated with organic findings

Clinical Manifestations —Glossopharyngeal neuralgia is characterized by spasms of excruciating lancinating *pain* which radiates from the *pharynx* and the *tonsillar fossae* to the *ear* The pain may be initiated by swallowing or yawning it lasts from a few seconds to a few moments and may recur many times Usually the attacks are intermittent and separated by long free intervals

Treatment —The treatment of glossopharyngeal neuralgia is less satisfactory than that of the trigeminal variety Paroxysms may be lessened by *cocainization* of the pharynx and tonsillar areas but alcohol injections cannot be accomplished In severe examples the expert neurosurgeon attempts *peripheral avulsion* which affords temporary relief *Intracranial division of the nerve* proximal to the ganglia, is necessary for a permanent amelioration of symptoms

GLOSSOPHARYNGEAL PARALYSIS

Isolated paralysis of the ninth nerve is not observed since the structure is never damaged without coincidental involvement of spinal accessory and vagus The symptoms and signs of glossopharyngeal involvement include *anesthesia* of the upper portion of the pharynx *loss of the sense of taste* over the posterior third of the tongue *difficulty in swallowing* and *abolition of the pharyngeal reflex*

VAGAL NEURITIS

The condition of *vagotonia* is discussed with the disturbances of the involuntary nervous system (p 1395)

VAGAL PARALYSIS

Vagal paralysis is an exceedingly *ominous circumstance* that is associated with inflammations scleroses tumors and vascular accidents involving the medulla Toxins such as alcohol lead and arsenic are said to influence this vital structure whose interruption results in unilateral paralysis of palate pharynx and larynx The voice assumes a nasal quality and difficulty is noted in swallowing Palatal and pharyngeal reflexes are absent and the corresponding vocal cord assumes the cadaveric position (p 2091) Disturbances occur in cardiac and respiratory rates and rhythms

PARALYSIS OF RECURRENT LARYNGEAL NERVE

Paralysis of the recurrent laryngeal nerve constitutes the most frequent injury to any division of the vagus This branch is quite vulnerable

and its function may be arrested following accidental severance during *operations on the thyroid* in *aneurysms of the aorta* in *enlargements of the left auricle* as in a tight mitrostenosis and in *inflammatory and neoplastic processes of the mediastinum*

The condition is suspected when the patient complains of *hoarseness* and the quality of the voice exhibits little change Examination of the larynx reveals the affected cord in a position midway between abduction and adduction (p 2091) There is no movement on phonation or respiration

Complete *bilateral recurrent laryngeal paralysis* produces total aphonia a lack of closure of the glottis in coughing difficulty in breathing respiratory stridor and dyspnea

PARALYSIS OF THE SUPERIOR LARYNGEAL NERVES

Isolated paralysis of the superior laryngeal nerves is infrequent it is associated with hoarseness a lowering of the pitch of the voice and a tendency of food to enter the larynx

Treatment —The treatment of paralyses of the laryngeal nerves calls for specialist consultation with the laryngologist (p 2025) Spasm of the glottis may require measures to prevent asphyxiation

NEURITIS OF THE SPINAL ACCESSORY NERVE

Paralysis of the eleventh cranial nerve may result from disturbances of the upper cervical region or the nerve trunk in the neck In the former instance the lesion may be a *poliomyelitis* a *progressive muscle atrophy* a *gliosis* or a *tumor* *Peripheral paralysis* results from caries of the upper cervical vertebrae neoplasms localized meningitis or injuries during operations on the cervical glands in the resection of a cervical rib or in association with a fracture of the base of the skull

Clinical Manifestations —Paralysis of the eleventh nerve produces motor disability of the *sternomastoid* and the *trapezius* As a result the patient is unable to turn the head toward the opposite side the shoulder droops and cannot be elevated the scapula is displaced downward and outward with external rotation

NEURITIS OF THE HYPOGLOSSAL NERVE

Central involvement of the twelfth nerve is a common accompaniment of the lesions that produce *hemiplegia* particularly the *vascular accidents* (p 1439) The nucleus becomes involved in *bulbar palsy* (p 1504) *syringobulbia* (p 1505) *tabes dorsalis* and diseases of the *medulla oblongata*

The *nerve trunk* may be injured in the posterior cranial fossa by tumors meningeal inflammations hemorrhages aneurysm of the vertebral artery fractures of the skull and inflammations of the bone

Clinical Manifestations —Hypoglossal paralysis is manifested by *deviation of the tongue* toward the paralyzed side This results from the unopposed action of the opposite genioglossus Speech is little affected by unilateral paralysis

PERIPHERAL MONONEUROPATHIES

The mononeuropathies that involve the peripheral nerves most often result from *local trauma* Irritations produce neuralgias or causalgias

TABLE 98 —PERIPHERAL NERVE INJURIES

Nerve Involvement	Common Etiologies	Motor Signs	Sensory Signs
Cervico occipital neuralgia	Focal infection Spheno ethmoiditis		Pain spasm and stiffness in regions of C 1–4
Phrenic nerve	The polyneuropathies (p 1499) Lead poisoning (p 76?) Alcoholism (p 3849) Diphtheria (p 302) Aneurysm of carotid Neoplasms or injuries in neck (p 3509) Aneurysm of aorta (p ,98) Mediastinal neoplasms Poliomyelitis (p 457) Neoplasms of cord (p 1430)	Dry nonproductive cough Intractable hiccough (p 1933) Ipsilateral paralysis of diaphragm	
Long thoracic nerve	Supraclavicular wounds and strains	Scapulae winged	
Suprascapular nerve	Blow on shoulder	Subluxation of humerus	
Axillary nerve (circumflex)	The polyneuropathies (p 1499) Fractures of the surgical neck of the humerus (p 3018)	Paralysis of deltoid *Inability to abduct the arms*	
Musculocutaneous	Fracture or dislocation of humerus	Paralysis of flexion of forearm Biceps reflex lost	Anesthesia of outer anterior surface of forearm
Radial nerve (musculospiral)	Compression during sleep Compression during operation Compression from splint during intravenous drip The polyneuropathies (p 1499) Plumbism (p 762) Trauma to forearm	Paralysis of extensor muscles of elbow Paralysis of triceps Wrist drop Finger drop	

Median nerve	Trauma to arm forearm or wrist	Weakness or absence of pronation of forearm Weakness or absence of radial flexion of the wrist Hand deviates to ulnar side Weakness or absence of flexion of metacarpophalangeal and phalangeal joints of the index and middle fingers Weakness or absence of flexion of the terminal phalanges of thumb Weakness or paralysis of abduction and opposition of thumb to fingers	Causalgia along sensory distribution with incomplete involvement
Ulnar nerve	The polyneuropathies (p 1499) Injury at the elbow or wrist	Paralysis of ulnar flexors of wrist Deviation of the wrist to radial side Paralysis of the flexors of the interphalangeal joints of the ring and little fingers Paralysis of the muscles of the hypothenar *eminence* Inability to abduct and flex the little finger Paralysis of the interossei and lumbricales Inability to adduct and abduct the fingers Inability to flex the metacarpophalangeal joint of the fingers Weakness of abduction of thumb	
Anterior crural (femoral) nerve	Diabetes mellitus (p 1210)	Thigh cannot be flexed on trunk leg cannot be extended Knee jerk lost	Anesthesia of inner side of thigh and leg
Intercostal neuralgia	Herpes zoster (p 435)		Girdle pain

TABLE 98.—PERIPHERAL NERVE INJURIES (*Continued*)

Nerve Involvement	Common Etiologies	Motor Signs	Sensory Signs
Lateral femoral cutaneous nerve	Tight corseting Fascial pressure Pes planus Obesity Cord tumor		Meralgia paraesthetica (pain in antero-external part of thigh) Anesthesia of region
Sciatic (extraspinal involvement) nerve	Local infection Local neoplasm Pre. ure from pelvic neoplasm Pressure from pelvic inflammation		Sciatica
Sciatic (extraspinal) nerve	*Osteoarthrosis of hip* *Dislocation of hip* Fracture of hip or thigh *Gluteal injection* Brodie's abscess (p 2982)		
Sciatic (intraspinal involvement) nerve	Herniation of the *intervertebral disk* (p 3074) Tuberculosis syphilis osteomyelitis or metastases in lumbar *and sacral vertebrae* Osteoarthrotic spurs of lumbosacral spine (p 2855) Back strain (pp 3063 3071) The polyneuropathies (p 1499) Arteriosclerosis of nutrient vessels	Paralysis of flexors of the knee *Paralysis of muscles below the knee* Inability to lift leg from the floor *Foot drag*	Sciatica

Common peroneal nerve (the external popliteal)	The polyneuropathies (p 1400) Injury to upper end of fibula Compression at upper end of fibula during sleep by bandages or through occupation Plumbism Arteriosclerosis of nutrient vessels	Atrophy and paralysis of anterior leg muscles Foot drop Loss of eversion and inversion of foot	
Tibial nerve (internal popliteal)	The polyneuropathies (p 1400)	Atrophy and paralysis of muscles of the calf and plantar muscles of foot Weakness or paralysis of extensors of ankle Weakness or paralysis of flexors of the foot Weakness or paralysis of the flexors of the toes Atrophic ulcer of heel	
Coccydynia	Trauma Conversion hysteria		Pain in coccyx
Metatarsalgia	Bad foot mechanics		Pain in 4th metatarsophalangeal articulation
Plantar neuralgia	Footstrain		Numbness and hyperesthesia in tips of toes and ball of great toe

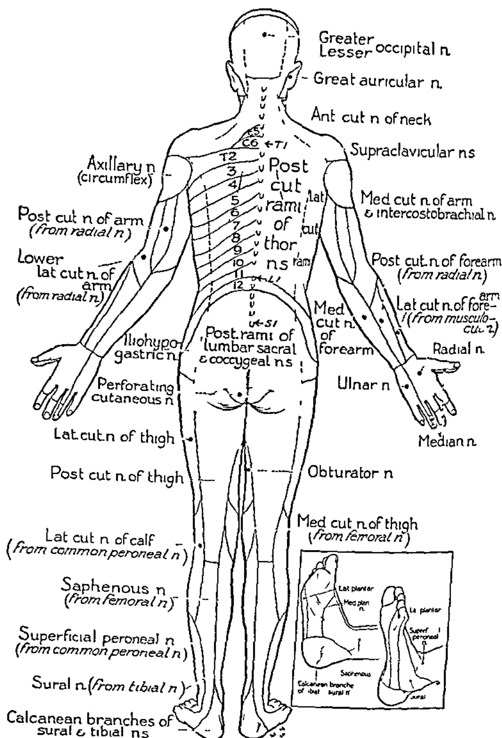


Fig 280—The cutaneous fields of peripheral nerves from the posterior aspect The boundaries of cutaneous supply of the posterior primary rami are indicated by broken lines The designation *Post cut rami of thor ns* refers to the cutaneous branches of the posterior primary rami *Lat cut rami* indicates the distribution from the lateral branches of the anterior primary rami For purposes of orientation the spinous processes of the first thoracic (T1) the first lumbar (L1) and the first sacral (S1) vertebrae are indicated by arrows

* Haymaker and Woodhall Peripheral Nerve Injuries

best illustrated by the *sciaticas* (p 3072) more complete injuries result in *paralyses* of motor nerves and *anesthesias* in the region of sensory structures

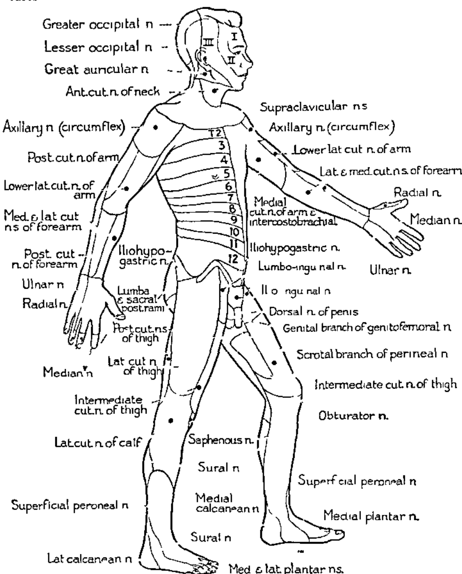


Fig 281.—Side view of the cutaneous fields of peripheral nerves The face and anterior half of the head are innervated by the three divisions of the trigeminal I ophthalm II maxillary III mandibular The fields of the intercostal nerves are indicated by numeral The unlabeled cutaneous field between great and second toe is supplied by the deep peroneal nerve

Mononeuropathies also arise as the result of an *elective localization* of a generalized systemic process Isolated musculospiral involvement producing wristdrop and footdrop from an affliction of the external popliteal

Haymaker and Woodhall, Peripheral Nerve Injuries

nerve may result from arsenical poisoning despite the absence of other demonstrable changes in the nervous system

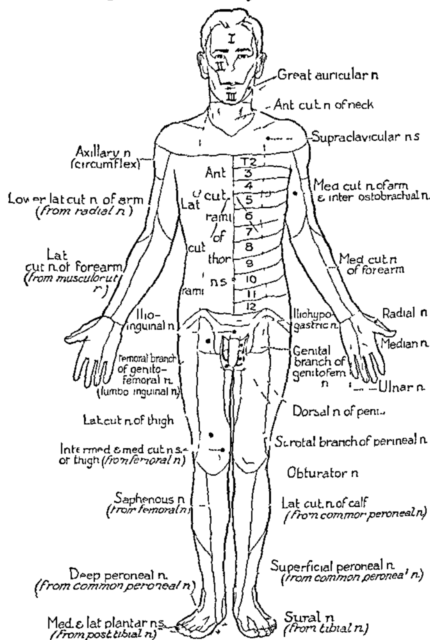


Fig 282—The cutaneous fields of peripheral nerves from the anterior aspect The numbers on the left side of the trunk refer to the intercostal nerves On the right side are shown the cutaneous fields of the lateral and medial branches of the anterior primary rami The asterisk just beneath the scrotum is in the field of the posterior cutaneous nerve of the thigh *

Clinical Manifestations—The clinical manifestations of the peripheral mononeuropathies are listed in Table 98 together with the common etiologies and the motor and sensory signs (p 1490)

Haymaker and Woodhall Peripheral Nerve Injuries

Diagnosis —The diagnostic investigation of a peripheral mononeuropathy differs in each instance *In general the local causes include* trauma congenital anomalies and the presence of local inflammatory processes or neoplasms The trauma may be *acute* as in the instance of a fracture or it may be *chronic* as in metatarsalgia The injury may be of recent origin or it may be long standing

The *local inflammatory processes* that cause impingement on a nerve are most often exudative articular processes particularly the *osteo-arthroses* (p 2855) In these the spurs press upon the trunks as they emerge from the vertebral column Less often the disturbance is caused by a *neoplasm*

Extraordinarily elective localizations are occasionally observed when a single nerve becomes affected in a systemic disturbance In the absence of definitive proof of local impingement the practitioner seeks more distant

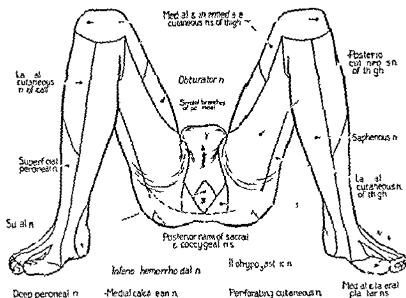


Fig 293 —Perineal view of the cutaneous fields of peripheral nerves

etiologic factors These include *focal infections* (p 42) or the occurrence of *neurotropic infections* such as herpes zoster and poliomyelitis *poisonings from chemicals* used in cosmetics or occupationally or by *drugs* administered for purposes of therapy *intoxications* resulting from metabolic diseases as *in the instance of diabetes mellitus or an avitaminosis*

Treatment —The treatment of mononeuropathy depends upon its causative agency *Local causes* are often corrected by appropriate surgery including adequate reduction of a fracture removal of exuberant callus division of an anomalous scalenus anticus or excision of the herniated nucleus pulposus *Offending drugs and chemicals* are eliminated and *endogenous intoxications* are ameliorated by controlling a disturbance such as a diabetes mellitus *Foci of infection* can be attacked by the dentist or the rhinologist

Haymaker and Woodhall Peripheral Nerve Injuries

TABLE 99.—DISTURBANCES OF THE PLEXUSES

Nerve Involvement	Common Etiologies	Motor Signs	Sensory Signs
Brachial plexus (upper radicular involvement)	Trauma to the supraclavicular space Fracture of clavicle Neoplasms of supraclavicular space Birth trauma (p 2951) See *Erb paralyses* *Klumpke paralyses*	Arm hangs limply at side Absence of abduction of arm Absence of lateral rotation of arm Absence of outward rotation of arm Absence of flexion of elbow Absence of biceps reflex	
Brachial plexus (lower radicular *involvement*)	Cervical rib *Scalenus anticus syndrome* (p 2959) Trauma to humerus *Forceful reduction of fractures of* humerus	Feeble radial pulse *Vasomotor changes and trophic changes in* skin of affected arm *Horner's syndrome* (p 1400)	Pain in shoulder and arm
Lumbosacral plexus	See *sciatic nerve* (p 1492)	See sciatic nerve and additionally Weakness and sensory involvement of anterior thigh and buttocks	
Cauda equina	See *cord tumors* (p 1430)		

Palliation—Palliative treatment of a mononeuropathy is often highly effective *Analgesics* are administered orally as acetylsalicylic acid or the salicylates Severe pains require additionally the use of an *opiate* preferably codeine Considerable comfort is afforded by the application of *heat* using an electric pad a hot water bottle hot compresses an infra red lamp or *diathermy* Enthusiasts for the last form of therapy claim specificity of results but our own experiences leave us in doubt whether this modality accomplishes more than any other form of heat (p 3788) We have no great confidence in *electrotherapy* whether by galvanism or faradism Paralyzed muscles are best prevented from stretching by the application of splints as in foot and wrist drop *Massage* can do no harm and is certainly of psychotherapeutic efficacy (p 3766)

Counterirritation is employed almost invariably with or without the consent of the physician *Oil of Wintergreen* (p 3122) is the most popular of the ingredients and its use has the recommendation of custom Intra venous injections of 100 to 200 mg of *thiamine chloride* are worthy of trial through the experience of their extraordinary efficacy in the management of tabetic crises (p 1467) Local *freezing* with ethyl chloride is sometimes helpful in herpes zoster

Surgery—Intractable pain requires more vigorous therapy Temporary relief is often afforded by injection or infiltration of trigger points with *procaine* for temporary effect and *alcohol* for more permanent relief Excruciating and intractable anguish may require the *intraspinal division* of the *sensory root*

POLYNEUROPATHIES

Polyneuropathy refers to the great group of peripheral nerve involvements with multiple manifestations As opposed to the mononeuropathies they are never due to local pathology but always result from systemic cause

Etiology—Despite the many different precipitating causes for polyneuropathy each agency eventually damages the nerve tissue by producing vitamin deficiency local ischemia or chemical destruction

See *Beriberi* (p 623) *Pellagra* (p 625)

The more common etiologic agents or precipitating factors include alcohol lead arsenic triorthocresyl phosphate mercury phosphorus carbon monoxide carbon disulfide benzene sulfur thallium acetate and sulfonamides Many of the disturbances attributed to injections of serums in fectious diseases blood dyscrasias metabolic disturbances and systemic disease of unknown etiology are probably specific vitamin deficiencies due to inadequate food intake inadequate gastro intestinal absorption or inadequate assimilation of food

Clinical Manifestations—Involvement of the nerves begins most frequently in the lower extremities with *pains* in the muscles of the calves and feet The discomfort may be sharp lancinating dull or boring with associated tenderness pins and needles sensations or burning of the soles toes and feet Coincident with preceding or following the *paresthesias* *weakness* of the feet and ankles is noted when small objects cause tripping and there is difficulty in walking in the dark

The pains may persist or be replaced by *numbness* The pain ascends higher in the limb while the burning of the soles of the feet continues

TABLE 99 —DISTURBANCES OF THE PLEXUSES

Nerve Involvement	Common Etiologies	Motor Signs	Sensory Signs
Brachial plexus (upper radicular involvement)	Trauma to the supraclavicular space Fracture of clavicle Neoplasms of supraclavicular space Birth trauma (p 2951) See *Erb paralyses* *Klumpke paralyses*	Arm hangs limply at side Absence of abduction of arm Absence of lateral rotation of arm Absence of outward rotation of arm Absence of flexion of elbow Absence of biceps reflex	
Brachial plexus (lower radicular involvement)	Cervical rib Scalenus anticus syndrome (p 2953) Trauma to humerus Forceful reduction of fractures of humerus	Feeble radial pulse Vasomotor changes and trophic changes in skin of affected arm Horner s syndrome (p 1400)	Pain in shoulder and arm
Lumbosacral plexus	See *sciatic nerve* (p 1492)	See sciatic nerve and additionally Weakness and sensory involvement of anterior thigh and buttocks	
Cauda equina	See *cord tumors* (p 1430)		

MYELOPATHIES

Toxic involvement of the spinal cord may be the result of the operation of those factors responsible for the peripheral neuropathies (p 1489) Manifestations are most commonly seen as the result of deficiency of niacin (p 620) as in *pellagra* (p 625) in *primary hyperchromic macrocytic anemia* (p 1077) with *poisonings* by carbon monoxide carbon sulfide and chloroform The traumatic myelopathies follow spinal and particularly caudal and epicaudal injections for anesthetic purposes

The clinical manifestations of any toxic myelopathy may be those of a degeneration of the *posterolateral columns* as in *tabes* (p 1464) there may be essential *pyramidal tract involvement* producing a spastic condition The presence of syphilis of the cord or a blood dyscrasia is eliminated by examinations of the hemogram the blood and spinal fluid serologies

Treatment requires the elimination of toxic agencies the administration of a high vitamin diet with accessory parenteral injections of all available preparations and the prevention of urinary sepsis

Caisson Disease —An almost specific involvement of the spinal cord seems to occur in caisson disease (diver s paralysis) Persons subjected to high atmospheric pressure as in diving or working under compressed air in a caisson develop these manifestations when they are suddenly returned to lowered or normal pressures On surfacing the rapidly decompressed worker complains of headache dizziness head pressure pains in the pit of the stomach (*the bends*) and pains in the limbs and back A complete or partial paralysis may develop involving both lower extremities The paraplegia is accompanied by varying degrees of sensory loss below the level of the lesion and there may be retention or incontinence of urine as in a transverse myelitis (p 1457)

Treatment requires prophylactic measures in that decompression is accomplished slowly and gradually

ENCEPHALOPATHIES

The encephalopathies are similar in their etiology to the polyneuropathies (p 1499) Most often they are due to *avitaminosis* or to chemical or pharmacological causation Common examples are seen in *alcoholism beriberi pellagra* and poisonings with *arsenic lead* or *mercury*

Clinical Manifestations —The clinical manifestations of a toxic encephalopathy are best illustrated in the experiences with *arsenotherapy* in syphilis (p 124) As a manifestation of sensitization rather than over dosage the patient becomes *irritable drowsy* and *confused* The symptoms may be mild and transitory or they may progress to *profound coma* or violent agitation with *convulsions* The *spinal fluid* is under increased tension but shows no other abnormality Autopsy fails to reveal the hemorrhagic manifestations that are said to exist in this condition and shows little change other than edema and round cell infiltration of the vessels

Treatment —The encephalopathy is best treated by *repeated lumbar drainage* and injections of 50 to 200 cc of 50 per cent *sucrose* Antidotal drug therapy is more apt to produce additional difficulty than amelioration of symptoms Injections of *sodium thiosulfate* and *cevitamic acid* have been of no value in our experience

The feet seem cold to the touch and are no longer dependable in recording the temperature of the bath The disability in walking is increased by involvement of the primary sensory modalities of *vibration* and *position sense* The same set of symptoms may appear in the hands and progress with the leg involvement As a rule there is no involvement of cranial nerves bladder or bowel

Neurologic examination reveals *flaccid atrophy* most marked in the feet and legs *impairment of the primary sensory modalities* greatest in the most terminal portions of the extremities, *hyperpathia* before there is complete loss of sensation and *trophic disturbances* most marked when the muscle and sensation are most involved

Certain of the specific neuropathies have characteristic clinical phenomena Those due to *alcohol* (p 1385) are often associated with bilateral footdrop and a resultant steppage gait resembling a *tabes* (p 1464) The *lead neuropathies* are often predominantly motor and associated with colic and encephalopathy (p 762) The palsy is often more prominent in the upper extremity and may be unilateral particularly causing wristdrop In *diphtheritic palsies* (p 302), the cranial nerve involvement is often predominant *Arsenical polyneuropathies* are prone to involve all four extremities and practically all muscle groups There may be an accompanying optic atrophy and encephalopathy ordinarily referred to as *hemorrhagic encephalitis* (p 124)

The polyneuritis of *thiamine chloride deficiency* (p 623), described as the clinical entity of *beriberi* is usually associated with a persistent edema and circulatory manifestations due to involvement of heart muscle The *pellagrous type of vitamin B deficiency* in addition to the peripheral neuropathy exhibits more marked evidences of degeneration of the posterior and lateral columns of the cord as seen in a combined degeneration (p 1502) The syndrome of *acrodynia* (erythredema polyneuritis) is probably a representation of vitamin deficiency as it occurs in infants and young children

Diagnosis —Polyneuropathy is such a clearcut reaction pattern that it can rarely be confused A search is required for the factor or factors which produce the etiological disturbances and their secondary reverberations in the central and peripheral nervous system

Treatment —The prime aim of therapy requires elimination of toxic factors and the immediate correction of the dietary deficiency by *adequate diet* and the use of *accessory vitamins* All of the vitamins are given since most of them are capable of producing changes in the central nervous system The vitamin intake of an adequate diet is often quite sufficient but if there is disturbance in gastro intestinal absorption the vitamins are given by parenteral injection using *thiamine chloride riboflavin* and *niacin* (p 620)

Physiotherapy is used for patients with wrist and footdrop Adequate splinting of the wrist is applied in the mid position and the use of splints or pillows at the bottom of the bed prevents stretching of the peroneal muscles *Massage* is given to maintain the tone of the muscle providing it does not induce pain If there is pain or tenderness of the muscles moist heat in the manner prescribed by Sister Kenny is used until pain and tenderness have disappeared

THE SCLEROSES

The scleroses constitute a baffling and discouraging group of neurological disorders Their etiologies are unknown and treatment is of no avail

SUBACUTE COMBINED SCLEROSIS

Subacute combined sclerosis is a *degenerative disease* of the spinal cord which usually begins after middle age It involves the *posterior horns posterior columns* and the *pyramidal tracts* The pathological manifestations were formerly seen much more frequently in the era that preceded liver therapy of *macrocytic hyperchromic anemia* (p 1077) There is a strong likelihood that present syndromes result also from metabolic causes

Clinical Manifestations —The symptoms and signs of subacute combined sclerosis are variable They may resemble a *tabetic syndrome* (p 1464) or appear as a *spastic monoplegia* or *paraplegia* *Painful paresthesias* are noted referable to the extremities The spinal fluid examinations are normal excluding *tabes dorsalis* blood counts and bone marrow studies reveal none of the phenomena of *hyperchromic macrocytic anemia* there is no satisfactory *therapeutic response* to the administrations of *liver and liver extracts* (p 1081)

Treatment —Treatment involves the elimination of any general hygienic error Therapeutic trials with *crude liver extract* or *bismuth* and *iodides* are warranted The suspicion of a cord tumor with or without positive spinal fluid findings (p 1434) warrants *exploratory laminectomy*

AMYOTROPHIC LATERAL SCLEROSIS

Amyotrophic lateral sclerosis *erroneously called chronic poliomyelitis* is a relatively rare disease It begins at the age of thirty five to forty and is more frequent in foreign born than in native Americans It is *chronic and progressive* and terminates fatally in *four to five years* The pathologic changes are confined to the *anterior horn cell groups* and the *pyramidal pathways* Degeneration of the gray matter in the motor strip of the brain cortex has also been found at autopsy

Clinical Manifestations —Amyotrophic lateral sclerosis usually is initiated by *symmetrical atrophy of the hands feet or tongue stiffness* and *weakness* Atrophy of the tongue is associated with difficulty of swallowing and chewing The involvement of muscles follows a bilateral symmetrical segmental distribution and the atrophy is associated with *spasticity hyperreflexia* and the appearance of pathological phenomena such as *clonus* and the *Babinski signs*

Course and Treatment —The course of the disease is that of progression until the *cranial nerve nuclei are involved with eventual fatal termination* It was thought that the administration of *vitamin E* (p 629) might arrest the progress of the disease but more objective studies have failed to show any specific therapeutic benefits

PRIMARY LATERAL SCLEROSIS

Primary lateral sclerosis *is an uncommon* disturbance of unknown etiology in which there is a *pure pyramidal tract degeneration* As a result the condition is characterized by gradually increasing *motor weak*

ACUTE SUPERIOR HEMORRHAGIC POLIOENCEPHALITIS (WERNICKE)

Wernicke's disease (acute superior hemorrhagic polioencephalitis) is probably due to *deficiency of thiamine chloride and niacin* (p 623) Often it is associated with *chronic alcoholism* (p 3851) which clouds the clinical manifestations

The onset of Wernicke s disease may be acute or gradual with head ache nausea vomiting insomnia or somnolence and diplopia Examina

TABLE 100 —THE SCLEROSES

Type	Age	Site of Pathologic Lesion	Clinical Manifestations
Subacute Combined Sclerosis	Middle age	Posterior horns posterior columns pyramidal tract	Spastic paralysis Tabetic syndrome
Amyotrophic Lateral Sclerosis	35–40	Anterior horns pyramidal tract	Atrophies especially of hands Spastic paralysis Bilateral and symmetrical involvement
Primary Lateral Sclerosis	Any age	Pyramidal tract	Spasticity Weakness
Progressive Bulbar Palsy	Older patients	Medulla Pons Bulb	Dysarthria Dysphagia Cranial nerve palsies Dysphonia Nasal voice
Multiple Disseminated Sclerosis	Young	Widespread with remissions and exacerbations	Diplopia Nystagmus Dysphonia (scanning speech) Ataxia Tremor Spastic Temporal pallor of optic disk
Syringomyelia	Any age	Central gliosis	Dissociation on sensory side with loss of pain and temperature contralaterally and touch anesthesia ipsilaterally Later pyramidal tract spasticity
Syringobulbia	Any age	Central gliosis	As above but with associated cranial nerve involvements and bulbar signs

tion reveals nystagmus partial or complete internal and external ophthalmoplegia rigidity of the extremities abnormal posture and abnormal movements suggesting disturbances in the basal ganglia The sleep rhythm is disturbed and there may be insomnia somnolence or coma

Wernicke s disease is best treated by intravenous injections of large doses of *thiamine chloride* and *niacin* Recovery takes place in several weeks but recurrences are to be expected if alcoholism continues

intravenous injections of histamine phosphate using 2 75 mg in 250 cc of isotonic saline at a rate of 2 0 to 6 0 cc per minute High vitamin *diets* are employed and accessory multivitamin preparations are administered No consistent beneficial effects have followed treatment with heparin (p 1050) dicoumarol (p 1049) hyperpyrexia (p 1379) quinine (p 516) liver extracts (p 1018) or deleading (p 764)

SYRINGOMYELIA

Syringomyelia is a degenerative disease of the spinal cord in which the predominant pathologic lesions are in or near the central canal (*status dysraphicus*) The lesion causes a disruption of function affecting the mid line structures of the body and central nervous system It may be that fundamentally there are *inclusion rests* along the line of closure of the neural tube and when these later proliferate and degenerate they produce local or diffuse degeneration and cyst formation in the cord and brain stem With syringomyelia there are often other associated midline *congenital defects* such as spina bifida improper closure of the cervical vertebrae supernumerary ribs and congenital anomalies of the hands feet and ears At times there is a proliferation of astrocytes about the central gliosis suggesting a new growth

Clinical Manifestations —The clinical manifestations of syringomyelia result from interruption of the crossing spinothalamic fibers There is diminution in the *pain* and *temperature* senses so that the patient develops *burns* and *trophic ulcerations* in the regions of the lesion *Morvan s disease* is a subvariety of syringomyelia in which there are ulcerations of the finger tips multiple paronychia and sensory changes With later progression there is *pyramidal tract involvement* with spastic weaknesses of legs and arms The sensory manifestations are contralateral whereas the motor disturbances are ipsilateral

Treatment —Syringomyelia is treated by *roentgen therapy* Occasionally evacuation of a single large intramedullary cyst is associated with some transitory improvement

SYRINGOBULBIA

Syringobulbia is a syndrome resembling syringomyelia (p 1505) except that there are involvements of the upper cord medulla and the cranial nerves Fibrillations atrophies and paralyses are noted in the tongue palate uvula and vocal cords Sensory changes are apparent in the regions innervated by the trigeminus with a crossed sensory dissociation

PARALYSIS AGITANS (SHAKING PALSY PARKINSON S DISEASE)

Paralysis agitans is a chronic affection of the central nervous system in which there is primary atrophy of the *striopallidal systems* Paralysis agitans has a definite familial and hereditary trend and is twice as common in men as in women It is characterized by *progressive weakness muscle rigidity* and *tremor* In older patients the disturbance is often on an *arteriosclerotic basis* but it may be neoplastic Juvenile and presenile types follow *epidemic encephalitis* (p 441) and may be associated with *exophthalmos* and signs of *hyperthyroidism* (p 1197)

ness spasticity exaggeration of tendon reflexes and a chronic course that may last many years Most often, the patient presents a *spastic paraplegia* with a 'scissors gait There are no sensory disturbances and pain does not occur

Treatment has been attempted surgically by division of posterior roots with inconsistent results

Progressive Bulbar Palsy

Progressive bulbar palsy is a rare disease of older people in which there are difficulties in speech swallowing mastication and phonation due to atrophy of the muscles of the lips tongue palate pharynx and larynx The disturbance is a degeneration of the cells of the *motor cranial nerves* situated in the *pons* and *medulla* It is probably the same disease as *amyotrophic lateral sclerosis* (p 1503) except for the site of the disturbance The etiology is unknown and pathological examinations show de*generations of the motor nuclei of XII XI, IX VII and V*

The *clinical manifestations* include difficulties in articulation in puckering the lips as in whistling and in speaking and swallowing The voice assumes a nasal character it is difficult to protrude the tongue which shows fibrillary tremors mastication and deglutition are impaired aspiration into the larynx may produce a pneumonitis The symptoms gradually increase for a few years after which death occurs from inanition There is no successful form of therapy

Multiple Sclerosis (Disseminated Sclerosis)

Multiple sclerosis represents a process of *demyelination* and seems to be increasingly prevalent Its etiology is unknown and the fundamental pathologic change seems to be a thrombosis of the local vessels causing areas of degeneration Present investigations are concerned with a possible relationship to *hemorrhagic diathesis* (p 1108) and the various forms of *endophlebitis*

Clinical Manifestations—Multiple sclerosis is a disease of *early adult life* and occurs equally in males and females Occasionally it seems to follow an acute infection The clinical manifestations of multiple sclerosis cover the entire range of neurology and psychiatry depending upon the area involved Frequent signs include transitory *double vision unsteadiness of an arm or leg tremor* and *disturbances of the bladder or bowel* with complete clearing in a few hours a few days or a few weeks The involvement is usually patchy and it is impossible to explain the findings on the basis of a single lesion such as might be present with a neoplasm (p 1430)

Course and Treatment—The course of the disease is characterized by *remissions* and *exacerbations* each acute episode is followed by some progressive secondary degeneration of the axis cylinder There is a steady increase in the amount of permanent damage to the nervous system with characteristic manifestations of pyramidal tract involvement causing *spasticity Disturbances of speech* are common as are *oculomotor palsies* and *nystagmus* The course of the disease is prolonged and the amount of incapacitation may be minor or major

Somewhat encouraging therapeutic results have followed repeated daily

often in sedentary and intellectual persons between the ages of sixteen and fifty The origin of the disturbance is unknown but precipitating causes are often clearly demonstrable

Aura —The migrainous headache is usually preceded by an aura This phenomenon has importance since it links the affliction to *epilepsy* (p 1515) and also serves as a herald manifestation to initiate preventive treatment (p 1508)

The aura though protean in its manifestations is usually distinct for each individual It may consist of irritability nervousness photophobia vomiting constipation an attack of diarrhea intestinal spasmosis scotomas hemianopsia lid lag paresthesias abdominal distention a sudden feeling of chilliness or flushing vertigo tremor sweating an aphasia or coldness of the extremities Sometimes the aura is recognized by the husband or wife who observes a change in disposition or a facial expression of tenseness Often the patient is not aware of the aura unless the physician urges concentration on the prodromal manifestations

Cephalalgia (Hemicrania) —The headache of migraine like the aura is usually individual It usually comes on at a *definite time* in the course of the day Morning afternoon and evening types are recognized Occasionally the headache bears a relationship to the *day of the week* Sunday headaches are observed in those who find difficulty in relaxing on the Sabbath Monday headaches are noted in those who overeat on the day of rest and have difficulty in overcoming their inertia for the beginning of the next week *Premenstrual menstrual postmenstrual* and *ovulation attacks* are observed in women who often note relief with the onset of menopause

The headache may be a complete hemicrania it may be orbital frontal temporal parietal occipital or referred down the neck and into the shoulders The description of the pain may be dull throbbing pulsating bursting or agonizing or there may be merely a feeling of fullness

Digestive Disturbances —The headache is almost invariably accompanied by gastro-intestinal manifestations such as anorexia a bad taste in the mouth a bad odor to the breath nausea vomiting spastic constipation or diarrhea Occasionally the gastro-intestinal symptoms are prodromal and the patient recognizes from the change in the character of the stool that a headache is imminent

Other Disturbances —At times the headache is associated with *ocular phenomena* such as a lid lag scintillating scotomas amaurosis hemianopsia or an ophthalmoplegia Photophobia is very common and few patients can stand exposure to bright or even normal light An *eosinophilia* occasionally is noted suggesting an *allergic element* in the disturbance (p 547)

Vasomotor phenomena usually accompany the headache The extremities become cold and clammy there may be flushing or blanching of the face and often the pulse rate slows perceptibly from vagal stimulation

Postmigrainous Euphoria —Following the migrainous headache there occur a sense of relief and a feeling of relaxation that borders on elation Cerebration is alert and crystal clear and this seems the 'best of possible worlds

Course —The course of the migrainous headache follows a unique and individual pattern It usually lasts for just so long and then disappears

Clinical Manifestations —The onset of paralysis agitans is insidious and the progress is slow and gradual The herald symptom is the appearance of a fine *rhythmic tremor* involving the index finger and thumb *Weakness* and *stiffness* are noted in the movements of the digits and soon the rigidity and adynamia increase in extent and severity until the entire body is involved

Tremor—The tremor is rhythmic, occurs at a rate of four to seven vibrations per second and has a characteristic *pill-rolling movement* It disappears during sleep and is brought out by voluntary movements best illustrated by having the patient attempt to drink a tumblerful of water

Rigidity—The rigidity is noted in the extremity musculature but also produces a *masklike facies* Winking is uncommon a generalized attitude of flexion is observed with the thumb turned into the palm the fingers flexed into a fist the neck depressed on the chest and the spine curved into a permanent kyphosis

Gait—A characteristic of paralysis agitans is the *propulsion gait* The steps are short and there is a tendency to *festination* until the patient finds himself running and can only stay his progress by bumping into an immovable object During walking the arms are held stiffly at the sides and they do not swing in the normal manner

Miscellany—Paralysis agitans is not associated with any significant changes in the reflexes sensorium or visceral functions There may be a slight *elevation in temperature* a tendency to *hyperidrosis* and *sialorrhea* Intelligence is not affected to the extent that might be anticipated by the stupid expression of the face

Treatment —The treatment of paralysis agitans is essentially symptomatic The patient may live for many years and die of some intercurrent condition The tremor may be well controlled by *belladonna* and similar preparations The oral use of *stramonium* (p 3875) in doses of 0 16 gm (2½ grains) four times daily has its adherents Drop doses of *tincture of belladonna* (p 3875) are recommended and emphasis has been placed on a *Bulgarian root* which is said to have greater potency than those of native habitat Commercial preparations such as *bellabulgara* and *rabellon* have had wide publicity, our best success has been with injections of *hyoscine hydrobromide* (p 3875) As little as 0.2 mg (1/300 grain), given subcutaneously may afford relief for several hours so that only two or three injections are needed daily for comparative comfort A member of the household may be entrusted to give the injections since self medication is prevented by the tremor

The combination of *belladonna* and *amphetamine sulfate* (benzedrine) seems particularly helpful in postencephalitic Parkinsonism especially in patients with oculogyric crises

MIGRAINE

Migraine is one of the varieties of *headache* (p 1510) Although the term often is used loosely as a synonym for headache it constitutes a definitive clinical entity that occurs only in a small per cent of the total number of cephalalgias

Clinical Manifestations —The typical migrainous headache is a *periodic hemicrania* Attacks are usually *familial* and *hereditary* They occur more

ANTI HISTAMINES —The successful introductions of anti histamines affords a fresh and hopeful approach to the management of migraine Both for prevention and therapy pyribenzamine (p 660) may be given in doses of 50 to 100 mg every two to four hours for at least six doses

OXYGEN THERAPY —Inhalations of oxygen give occasional relief to the *migrainous headache The method is worthy of trial if a tank and a mask* are available

ERGOTAMINE —Concurrently with the use of the analgesic *ergotamine tartrate* marketed as *Gynergen* (p 3833) or dihydro ergotamine is given subcutaneously in a dose of 1 mg (1/60 grain) or intravenously in the amount of 0 4 mg (1/160 grain) The initial injection of Gynergen is given by the physician at his office The patient remains under observation for at least an hour to note any *idiosyncrasy* such as nausea vomiting paresthesias and angiospasms of the extremities The drug is withheld in pregnancy in patients with significant arteriosclerosis hypertension angina or Raynaud s disease

In the absence of untoward manifestations the patient is taught to self administer the drug by hypodermic and the dose is regulated according to the individual needs

THIAMINE CHLORIDE —The treatment of migraine has been advanced also by the use of intravenous injections of 100 to 200 mg (1½ to 3 grains) of *thiamine chloride* (p 622) These are given in addition to the headache remedy and the Gynergen

MISCELLANY —An occasional patient reports relief by the use of *pressure* to trigger points such as the regions of the temporal or occipital foramina These maneuvers emphasize the possible pathogenesis of a migrainous headache as a distention of the relaxed walls of the cranial arteries An *ice cap* to the head is often grateful though some patients prefer *heat* A *scalp rub* with 0.5 per cent *menthol* in alcohol or with a *menthol stick* (headache pencil) sometimes gives a measure of comfort

Other *infrequently applied and not consistently useful agents include* injections of hypertonic salt solutions the pituitary extracts or caffeine Inhalations of *nitroglycerine* have been advised but our experiences emphasize the greater likelihood of increase in the headache after temporary relief Attempted vasoconstriction by the uses of *ephedrine* or *amphetamine* (benzedrine) has met with no consistent benefits in our experiences

Injections of potentially habit forming drugs are reserved for dire occasions Demerol codeine dilaudid and morphine are withheld if it is at all possible

Interval Treatment —After an attack most patients are eager for prophylactic therapy of any sort General instruction is given in *personal hygiene* with particular reference to working hours reading habits the use of the eyes the care of the bowels an abundance of outdoor exercise and longer hours of sleep Often a *holiday* is followed by considerable surcease (p 3761) The *nasal accessory sinuses* are investigated and any inflammatory process is eradicated if possible The *teeth* are carefully examined and possible foci of infection are eliminated There is always a vogue for *colon irrigations* and many patients testify to their usefulness A *saline purge* taken each Sunday probably fulfils the same purpose at lesser trouble and expense

The duration may be several hours or several days Repetitions may be frequent or unusual An associated *epilepsy* (p 1515) occurs in a ratio that is disproportionately high in reference to the general population

Treatment—In the management of migraine the aim of the therapeutist is the prevention of the recurrence of attacks In the event of failure, the individual headache requires symptomatic attention

Prophylaxis—Since each migrainous sufferer presents a unique problem it is good practice to request the patient to keep a 'diary history' (p 3474) Many interesting clues are obtained which have important bearing on the prevention of attacks Certain of the more common *precipitating circumstances* include emotional strain constipation, eating before bedtime exposure to special foods such as milk chocolate eggs pork nuts cauliflower tomato, or cabbage excessive smoking, the particular use of cigar or pipe a delayed bowel movement, fatigue alteration in the daily routine such as eating the heavy meal in the middle of the day on Sunday excessive use of alcohol in any form or of some particular brand such as gin rum brandy, or a mixture of drinks, menstruation or ovulation, excessive indulgence in sexual intercourse, prolonged periods of sexual continence eyestrain due to reading in bed until the late hours of the night, imperfect illumination and excessive purging

The study of the diary assists greatly in spacing out and perhaps preventing the onset of the migraine It has value too in the attempt to abort an attack, upon recognition of the aura

Abortive Treatment—The use of the diary history enables the patient to recognize the individual pattern of the aura The value of active treatment during this period far transcends that of measures employed once the headache has begun Perhaps the greatest single effectual effort is *evacuation of the bowels* by the use of an *enema* or a *saline purge* Each sufferer from migraine agrees on the value of rest in a darkened room If it is at all convenient a *warm relaxation bath* is taken and the affluent find comfort in *massage* (p 3766) The oral use of *calcium lactate* in doses of 2 gm (30 grains) every two hours and inhalations of 100 per cent *oxygen* are successfully employed by some sufferers Active treatment with drugs is vigorously initiated, as outlined in the next paragraph rather than procrastination until the headache begins

Active Treatment—If abortive treatment has not been initiated the measures previously described are started at the onset of the headache

THE HEADACHE CAPSULE'—The headache capsule is taken orally and washed down with a *caffeinated beverage* such as coffee tea or a cola drink

℞	Acetylsalicylic acid	5 0
	Acetphenetidin	4 75
	Phenobarbital	0 25
	Divide	
	Ft Caps or Powders No 15	
	Sig.. One hourly for 3 doses	

or

℞	Acetylsalicylic acid	4.5
	Codeine phosphate	0.5
	Divide	
	Ft Caps No 15	
	Sig One hourly for 3 doses	

insensitive to pain Only in the vicinity of large vessels are *dura* and *arachnoid* responsive to painful stimuli Thus the actual site for the production of the sensation of pain in the head is narrowed to the vessels which may be altered by vasoconstriction vasodilatation or stretching

The importance of vascular elements in the pathogenesis of headache is emphasized by the effects noted with drugs whose action is on the peripheral vascular musculature Thus headache may be produced by *epinephrine* which elevates blood pressure through vasoconstriction and by *nitrites* and *histamine* which rapidly lower vascular tension by producing widespread vasodilatation

Clinical experience points to the importance of disturbances in spinal fluid pressure relationships Lumbar puncture with withdrawal of spinal fluid is often but not invariably associated with headache Headache also occurs when air is injected into the ventricles in the course of diagnostic pneumoencephalography

Despite the production of headache by disturbances in pressure relationships the majority of complaints as ordinarily seen are unaccompanied by definite alteration The practitioner is therefore reduced to interpreting the derangement as a manifestation of toxemia Some of the toxic processes are capable of tangible definition as in the instance of headaches that accompany anoxemia acute anemia acidosis dehydration and water intoxication In certain examples of migraine occurring near the time of the menses there seems to be an excess of gonadotropic hormone in the urine with a deficiency of estrogen suggesting a hormonal imbalance as the provocative factor in the disturbance

In the majority of clinical experiences unfortunately the toxic factor is not demonstrable Those who enjoy speculation concerning medical problems hypothesize the release of histamine like substances from the bowel or of internal urticarial lesions within the confines of the cranium

Types of Headache and Their Management —The management of acute or recurrent headache may be facilitated by attention to the following details regarding the type of headache

1 —*Frontal and supra-orbital headache* is more common with sinusitis intranasal disturbances and *trigeminal neuralgia* involving the ophthalmic branch

2 —*Unilateral temporal headaches* suggest migraine or temporal arteritis They also occur with disturbances of the eye and ear

3 —*Facial pain* may be due to involvement of the antra or of the trigeminal or sphenopalatine nerve elements

4 —*Occipital pain* occurs in spheno ethmoiditis hypertension eye strain chronic nephritis meningitis indurated myositis (nodular headache) and cervical occipital neuralgia

5 —The *vertex headache* is probably hysterical

6 —*Pounding headache* occurs more often with migraine than hypertension

7 —The etiology of *recurrent headache* is often best elucidated by the diary history with particular emphasis upon overeating over drinking oversmoking excessive indulgence in candy chocolate or starches constipation fatigue mental or emotional strain or stress insomnia excessive use of coffee or tea poor

Those who favor the allergic aspect of migraine practice *desensitization injections* of *proteoses* and *peptones* Multiple *skin tests* are performed and suspicious substances are forbidden in the diet *Elimination regimens* (p 562) are often utilized with some measure of success The students of metabolism advocate the use of *ketogenic diets* (p 675) but these are impracticable for prolonged use Probably an analogous effect may be obtained with less nuisance by administering 6 to 8 gm of *ammonium chloride* daily for three to four days at the expected time of the headache Pyribenzamine (p 565) should be continued intermittently

Enthusiasts for *hormone therapy* point to the increased excretion of *prolan* in the urine at the time of the headache and to the relationship that is often demonstrable between ovulation menstrual cycle and attacks We are not averse to injections of *anterior pituitary extracts* (p 1154) for a limited probatory period, but we entertain a healthy respect for the side effects which may be associated with the uses of *androgen* and *estrogen* The former is capable of resulting in sexual impotence in the male and may postpone menstruation and cause the appearance of male secondary sexual characteristics in the female (p 2515) Estrogen has carcinogenic potentialities which menace the peace of mind of conservative practitioners such as the senior author

Useless Measures—The long list of useless measures includes desensitization with histamine the administration of histaminase (torantil), Veratrum viride Gelsemium Cannabis indica and surgical procedures such as tonsillectomy appendectomy cholecystectomy corrections of uterine malposition perineal repairs resections of the large bowel or the roots of the fifth cranial nerve

HISTAMINE CEPHALALGIA

Histamine cephalalgia is thought to be a form of unilateral headache that is distinct from migraine It is of brief duration and is associated with *tearing* and *vasomotor rhinitis* on the affected side It is reproduced by injections of *histamine* and may be controlled by anti histamine drugs particularly pyribenzamine (p 565) in doses of 50 to 100 mg

THE GENERAL MANAGEMENT OF HEADACHE

Headache is a common presenting symptom For every one concerning which the physician is consulted however countless headaches occur for which the patient practices self medication with popularly advertised home remedies

The Mechanism of Headache.—Despite the frequent occurrence of headache there is limited knowledge concerning its mechanisms Clinical data relate to observations concerning the sensitivity of cranial and intracranial structures as observed under local anesthesia the effect of drugs which alter blood pressure the results of alterations in spinal fluid pressure relationships and problems of toxemia

The neurosurgeon operating under local anesthesia notes that dura arachnoid pia cerebral tissues choroid and ventricular ependyma are

insensitive to pain Only in the vicinity of large vessels are *dura* and *arachnoid* responsive to painful stimuli Thus the actual site for the pro duction *of the sensation of pain in the head is narrowed to the vessels* which may be altered by vasoconstriction vasodilatation or stretching

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DIFFERENTIAL DIAGNOSIS OF

Headache

Lacking a clearcut conception of the toxic factor operative in most cases of headache and of the mechanisms by which noxious elements operate the practitioner is dependent upon his clinical acumen in elucidating the problems of headache In the majority of instances, his great reliance is placed upon the accuracy and care with which he collects and evaluates the material of the history and the thoroughness with which he undertakes the physical examination

CAUSE	DIAGNOSTIC FEATURES
Errors in Hygiene	Excitement anxiety constipation gluttony excessive fatigue indulgence in alcohol overheating insolation, excessive smoking Idiosyncrasy to chocolates starch, candy or eggs. The wearing of a tight hat band Poor ventilation insomnia excessive noise overbright or poor illumination Check history
Pharmacologic	From epinephrine nitrite histamine caffeine opiate general anesthetics hypnotics iodides, salicylates quinine quinidine or sulfonamides Following spinal or caudal anesthesia Check history
Toxic	Overdoses of the drugs above mentioned Fumes in industry Jaundice
Metabolic	Anoxemia dehydration acidosis cholemia, premenstrual or menstrual tension, renal insufficiency azotemia methemoglobinemia, hypoglycemia fever allergies hyperpituitarism and auto-intoxication Urinalysis and blood chemistry (p 3712)
Hemic	Anemia and polycythemia Obtain hemogram (p 3704)
Circulatory (Systemic)	Hypertension and hypotension Particularly with hypertensive toxemia of pregnancy and malignant phase of hypertension (p 916) With forward and backward failure (p 941)
Neurocirculatory	Angiospasms Cerebral arteriosclerosis embolization or thrombosis With increased intracranial tension In temporal arteritis With cerebral accidents such as rupture of an aneurysm of the circle of Willis and subarachnoid or interventricular hemorrhage With edema of the brain
Infections (Generalized)	Particularly with influenza typhoid, typhus spotted fever tuberculosis brucellosis, secondary syphilis relapsing fevers dengue measles meningococcemia yellow fever infectious jaundice malaria and infectious mononucleosis
Infections (Neurologic)	Nonsuppurative encephalitides Brain abscess Meningitides Poliomyelitis Check spinal fluid (p 3734) Temporal arteritis (p 1037)
Neuroses	With anxieties hysteria (clavus) neurasthenia and hypochondriasis Malingering
Psychoses	General paresis and cerebrospinal syphilis Check spinal fluid (p 3734)

Neurogenic	Migraine (p 1506) Epilepsy (p 1515) Trigeminal sphenopalatine or occipital neuralgia (p 1482) Hydrocephalus (p 1409) Get x rays electrocardiogram, and check spinal fluid
Osseous	Osteomyelitis and gummas of the skull Following trauma concussion or fracture With osteitis deformans, multiple myeloma and skeletal metastases Get x rays
Skeletal	Cervical spondylitis and occipital myositis or fibrositis Take x rays of spine
Respiratory	With upper respiratory infection, nasal obstruction, pansinusitis spheno-ethmoiditis and Sluder s syndrome Get x rays of sinuses Refer to specialist for lavage of antrum and cocainization of ganglion
Ophthalmic	With eye strain, conjunctivitis iritis keratitis retinitis orbital cellulitis and glaucoma Refer to specialist
Otogenic	With otitis media, labyrinthitis mastoiditis and petrositis Refer to specialist

ventilation in the bedroom or the workshop the inhalation of noxious fumes in industry eye strain excessive noise or lighting insufficient illumination the onset of menstruation the habitual use of drugs such as narcotics hypnotics analgesics and sulfonamides exposure to sun

8 —A *band headache* usually characterizes fatigue

9 —A *hammering headache* is often of hysterical origin

10 —The headache that is *worse in the morning* is often due to sinusitis or alcoholism

11 —The headache that *is worse at night is often due to intracranial* disease osteomyelitis of the skull nephritis or eye strain

12 —*Recurrent headaches with a definite pattern* are almost invariably of migrainous origin

13 —The *Sunday morning headache* is either migrainous or a hang over of the Saturday night debauch

14 —The *Monday morning headache* is usually due to malingering

15 —*Cyclic headaches* which occur intermenstrually premenstrually or menstrually are often due to disturbances of water metabolism and are relieved by ammonium chloride diuresis (p 614)

16 —*Relief* of headache following an *enema* or a *purge* suggests the factor of auto intoxication

17 —*Relief* of headache following an *analgesic antipyretic* such as acetosalicylic acid 0 3 gm (5 grains) suggests that the headache is of casual and minor importance

18 —*Relief* of headache following the injection of 0 5 to 1 mg (1/120 to 1/60 grain) of *ergotamine tartrate* suggests that the headache is of migrainous origin

19 —*Exacerbation* of headache when the *head is bent* over or *shaken* suggests sinusitis

20 —In *febrile headache of recent origin* the nose nasal accessory sinuses and spinal fluid warrant investigation

21 —With headache in *association with eye or ear disorders* specialist consultation is mandatory

22 —Except for the headache of the hypertensive toxemia of pregnancy and malignant hypertension in the young and middle aged *elevation of blood pressure* is usually an accompaniment and not a cause of headache

23 —Except in marked degrees of hypotension *lowering of blood pressure* is usually an accompaniment and not a cause of headache

24 —Except in profound or acute anemia a *blood disturbance* is rarely a cause of headache

25 —Except in marked polycythemia a blood disturbance is usually an accompaniment and not a cause for headache

26 —*Neuralgias* of the *trigeminus* and *sphenopalatine elements* yield to local cocainization or alcohol injection

27 —Headache *accompanied by flushing of the face lacrimation* and *acute swelling of the nasal mucous membrane* occurs with the histamine variety of the complaint

28 —*Skull tenderness* may be demonstrated over the *brain abscess or* brain tumor

29 —*Sinus tenderness* is particularly acute in recent infections

30 —The *points of emergence* of the branches of the trigeminal nerve are exquisitely tender in the neuralgias

31 —*Mastoid tenderness* is of grave significance in ear infection

32 —Thickening of the insertion of the heavy muscles into the occiput suggests the *nodular type of headache* due to an indurative myositis

33 —Lavage of the nasal accessory sinuses and radiographs are indicated if there is *obstruction to the airway nasal* or *post nasal discharge* a *history of recent cold congestion or swelling of the turbinates nasal polyps* or deficiencies in *transillumination*

34 —*Hardening of the eye balls* with intense headache suggests glaucoma and calls for the prompt use of miotics (p 1548)

35 —*Examination of the fundus oculi* should be a routine procedure in the investigation of any headache

36 —*Inspection of the ear drums* should be routine in unilateral headaches particularly in infancy and childhood

37 —With febrile headache *rigidity of the neck* should be sought Lumbar puncture is performed if there is any suspicion of difficulty

38 —A *routine urinalysis* is required in the investigation of all headaches Pertinent findings include fixation of the specific gravity albuminuria glycosuria, acetonuria and indicanuria

39 —A *routine blood count* is required in the investigation of the more persistent and severe grades of headache Anemia polycythemia and leukocytosis merit further investigation

40 —*Pertinent blood chemical findings* include hypoglycemia hyperglycemia azotemia cholemia and methemoglobinemia

41 —A *neurological status examination* is required in all persistent or recurrent headaches

42 —*Psychiatric investigation* is merited if there is a suspicion of hysteria

43 —*X-ray of the skull* is necessary if there is any history of preceding trauma or any compensation factor

44 —*Indurative headache* is often relieved by local massage

EPILEPSY

It is conservatively estimated that there are one half million epileptics in the United States The syndrome of *idiopathic epilepsy* which is differentiated from *symptomatic epilepsy* such as occurs with brain tumor is best described as a paroxysmal cerebral dysrhythmia

Etiology —Epilepsy is a *familial disease* in 20 per cent of patients It may be related to birth traumas intracranial pathology and migraine *Like the latter it is probably a metabolic disorder with the sudden liberation of some endogenous toxin*

The Epileptic Constitution —The epileptic constitution is clearly recognized Most patients are gloomy easily irritated suspicious emotionally impoverished as best illustrated by the monotony of their conversation and their conversational tone quarrelsome hypersensitive subject to violent rages somewhat paranoic sulky and disagreeable It is difficult to *determine how much is part of the epilepsy and what portion results* from the knowledge and fear of the seizures

Many epileptics remain mentally normal Others deteriorate partially as the result of the disease but mostly from the injudicious use of bromides and alcohol and the psychological implications of their affliction

Clinical Manifestations —The clinical manifestations of epilepsy are characterized by an *aura* a *convulsive stage* (*grand mal*) and a *postepileptic phase* The convulsive stage may be replaced by a *petit mal* or a *psychic equivalent*

The Aura —The aura of epilepsy like that of migraine is individual and characteristic It may consist of some optic olfactory gustatory motor sensory emotional or characterological disturbance The observant patient *will almost invariably recognize the prodrome which may be apparent also* to a mother a husband or a wife

Convulsion —The epileptic convulsion is characterized by *loss of consciousness* and a *falling to the ground* There is often an eerie and ominous *cry* The convulsion is *tonic* at first but later becomes *clonic* and is often associated with *involuntary urination* and/or *defecation* At times there is *erection* with *ejaculation* in males

During the convulsive period the face becomes ashen and then cyanotic due to breath holding The tongue is bitten and there is foaming at the mouth The pupils are usually miotic at first but later they become mydriatic During the tonic and clonic phases respiration may be slowed or a period of complete apnea is observed At this time the pulse becomes almost imperceptible

Petit Mal —The convulsive form of grand mal of epilepsy is sometimes replaced by petit mal characterized by transitory *loss of consciousness* The attack may be so fleeting that the patient is unaware of its existence Observers note a blankness of expression a rolling of the eyes or a sudden *drooping of the head The patient has an amnesia* during the period of attack and goes on with conversation and activity as if nothing had happened These attacks of petit mal are particularly dangerous since the sub

ject may suffer injury to himself during the period of unconsciousness or he may inflict grave damage to others if, for example he is driving a motor car or working with machinery

Psychic Equivalents—Psychic equivalents occur in epileptics in place of the attacks of grand or petit mal The individual becomes manic irate homicidal exhibitionistic or perverse In children, the affliction may be mistaken for inexplicable naughtiness At times the equivalent is accompanied by muscle twitching blinking word perseveration or other forms of automatism (p 1308) Associated attacks of migraine occur with undue frequency

Status Epilepticus—The most violent type of epilepsy is that in which there are rapidly recurring attacks (status epilepticus) These may be accompanied by stupor, hyperpyrexia circulatory failure coma and even death

Postepileptic Phenomena—Following the grand mal particularly post epileptic phenomena are noted They may consist of stupor sleep automatism amnesia and particularly headache Some patients become aware of an attack only through the development of cephalalgia or the appearance of a bitten tongue Following epilepsy there may be a period of automatism in which irrational and even criminal acts of violence are performed The latter have medicolegal significance

Diagnosis—The diagnosis of epilepsy was formerly based on clinical manifestations The introduction of *electro encephalography* (p 1403) permits curves to be presented illustrating grand and petit mal and the psychic equivalents When possible the practitioner should refer his patient to the expert for this information in the manner in which he obtains an electrocardiogram in the patient with circulatory disease although all epileptics do not show abnormal graphs

Despite electro encephalography the diagnosis of idiopathic epilepsy is best made by exclusion A careful *neurologic status* is conducted relative to motor sensory and reflex phenomena *A film of the skull* is required and the *spinal fluid* is examined cytologically serologically and by the colloidal gold curves The *optic nerve head* is viewed by the ophthalmoscope and the *visual fields* are plotted to detect defects

When there is doubt concerning the diagnosis of epilepsy the water pitressin test may be employed Anticonvulsive medication is avoided for twenty-four hours prior to the test The patient is kept in bed on a normal diet throughout the test and for an additional twenty four hours, at hourly intervals when the test is started a pint of water is ingested starting with the fourth pint of water pituitrin is given hourly in doses of 0 2 0.3 0 4 0 5 0.5 0.5 0.5 and 0 5 cc the fluid intake and output are charted and the blood pressure is recorded hourly

In the positive test a convulsion is induced in more than 40 per cent of those who suffer from true epilepsy The test is discontinued with the appearance of severe headache gastro intestinal distress elevation of blood pressure or the actual convulsion

The presence of any positive finding demands specialist consultation for an investigation of organic intracranial disease such as neoplasm infection a vascular accident or an unsuspected fracture Pneumencephalography is required when focal signs or focal seizures are present

Treatment—The treatment of epilepsy follows the plan employed in migraine (p 1508) *A diary is kept to reveal precipitating circumstances* Efforts are made to prevent the seizure since little can be done once the attack has started

Prevention—Attempts are made to prevent attacks of epilepsy by general measures of hygiene *The errors that must be avoided include over* eating overdrinking fatigue emotional disturbances constipation excessive sexual intercourse excessively prolonged sexual continence and the abuse of alcohol or tobacco

DIETOTHERAPY—*Prophylactic dietotherapy in the management of epil*epsy consists of the use of a *ketogenic routine* (p 675) This is distinctly an institutional procedure and of temporary value in patients who are having repeated or severe attacks Perhaps the same purpose can be accomplished by the generous administration of acidifying salts such as *ammonium chloride* 6 to 8 gm daily for three days on and two days off

PHARMACOTHERAPY—Useful drugs include tridione phenobarbital and dilantin Tridione (3 5 5 trimethyloxazolidine 2 4 dione) is dispensed in capsules containing 0.32 gm (5 grains) Its daily dose is from 1 0 to 2 0 gm irrespective of age Tridione is of particular value in treating seizures with divergent clinical appearances pykno epilepsy and myoclonic and akinetic attacks Aplastic anemia and agranulocytosis have followed tridione administration

Many epileptics are quite completely controlled by *phenobarbital* given in amounts of 0 06 to 0 13 gm (1 to 2 grains) daily Except for a rare eruption phenobarbital has no significant untoward effects and the results have been quite satisfactory Dilantin given in doses of 0 1 gm (1½ grains) three times daily has proven even more effective than phenobarbital with which it can be alternated or combined Dilantin has the disadvantage of causing more frequent untoward symptoms such as nausea vomiting substernal burning bleeding of the gums stiffness tremor *ataxia and a generalized eruption The hemorrhagic tendency may be pre*vented by the simultaneous administration of *cevitamic acid* (p 629) Unlike phenobarbital dilantin does not cause any depression and has proven a tremendous addition to the armamentarium of epilepsy

More recent innovations in the therapy of epilepsy are glutamic acid and mebaral Mebaral known in Europe as prominal is methylethyl phenylbarbituric acid It is dispensed in tablets of 0 2 gm (3 grains) and the average dose is 1 tablet two or three times daily Mebaral may be substituted for phenobarbital but the changeover should be gradual increasing the dose of the mebaral as the phenobarbital dose is decreased and then discontinued

COMBINATION THERAPY—A combination form of prophylactic therapy in epilepsy is suggested as undernoted

1 Accomplish *dehydration* once weekly by limitation of fluids to 600 cc and the use of a saline purge
2 Produce a therapeutic acidosis two or three days weekly by administering *ammonium or calcium chloride* in doses of 6 to 8 gm using capsules containing 0 5 gm
3 For the first half of each month give *phenobarbital* in the dosage of 0 06 to 0 13 mg (1 to 2 grains) daily and during the last half

ject may suffer injury to himself during the period of unconsciousness or he may inflict grave damage to others if for example he is driving a motor car or working with machinery

Psychic Equivalents—Psychic equivalents occur in epileptics in place of the attacks of grand or petit mal The individual becomes manic irate homicidal exhibitionistic or perverse In children the affliction may be mistaken for inexplicable naughtiness At times the equivalent is accompanied by muscle twitching blinking word perseveration or other forms of automatism (p 1308) Associated attacks of migraine occur with undue frequency

Status Epilepticus—The most violent type of epilepsy is that in which there are rapidly recurring attacks (status epilepticus) These may be accompanied by stupor hyperpyrexia, circulatory failure coma, and even death

Postepileptic Phenomena—Following the grand mal particularly, postepileptic phenomena are noted They may consist of stupor sleep automatism amnesia and particularly headache Some patients become aware of an attack only through the development of cephalalgia or the appearance of a bitten tongue Following epilepsy there may be a period of automatism in which irrational and even criminal acts of violence are performed The latter have medicolegal significance

Diagnosis—The diagnosis of epilepsy was formerly based on clinical manifestations The introduction of *electro encephalography* (p 1403) permits curves to be presented illustrating grand and petit mal and the psychic equivalents When possible the practitioner should refer his patient to the expert for this information in the manner in which he obtains an electrocardiogram in the patient with circulatory disease although all epileptics do not show abnormal graphs

Despite electro encephalography the diagnosis of idiopathic epilepsy is best made by exclusion A careful *neurologic status* is conducted relative to motor sensory and reflex phenomena *A film of the skull* is required and the *spinal fluid* is examined cytologically serologically and by the colloidal gold curves The *optic nerve head* is viewed by the ophthalmoscope and the *visual fields* are plotted to detect defects

When there is doubt concerning the diagnosis of epilepsy, the *water pitressin test may be employed Anticonvulsive medication* is avoided for twenty four hours prior to the test The patient is kept in bed on a normal diet throughout the test and for an additional twenty four hours, at hourly intervals when the test is started a pint of water is ingested starting with the fourth pint of water pituitrin is given hourly in doses of 0.2 0.3 0 4 0.5 0.5 0.5 0 5 and 0 5 cc the fluid intake and output are charted and the blood pressure is recorded hourly

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are *paroxysmal* are described as *clonic* whereas maintained movements are of a *tonic* character

Treatment of Convulsions —It is the obligation of the practitioner to protect the patient during the convulsive seizure Afterward diagnostic in

DIFFERENTIAL DIAGNOSIS OF

Convulsions

Generalized convulsions have less diagnostic significance than local or Jacksonian seizures which indicate a fairly localized lesion. Most convulsions are associated with unconsciousness, amnesia and loss of sphincteric control The most frequent cause for repeated convulsive episodes is idiopathic epilepsy

CAUSE	DIAGNOSTIC FEATURES
Infectious	Nonspecific manifestation of acute invasion particularly in infancy and childhood. With encephalitides (p 1463) meningitides (p 1462) and tetanus (p 294) Check spinal fluid.
Metabolic	Hyperpyrexia particularly in infancy and childhood. Tetany and rickets. Alkalosis and hypocalcemia Diabetic coma and hypoglycemia Uremia, azotemia and renal insufficiency Cholemia Anoxemia and asphyxia Addisonian crises (p 1275) Obtain urine and check blood chemistry
Neurogenic	*Epilepsy* (p 1519) Following trauma particularly to the skull With local infection such as encephalitis poliomyelitis meningitis rabies tuberculous meningitis general paresis syphilitic meningo-encephalitis brain abscess and lateral sinus thrombosis (p 1446) With hydrocephalus tuberous sclerosis and idiocy With brain neoplasms and cysts Supplement neurologic examination with x rays, electrocardiogram, spinal fluid examinations and specialist consultation.
Psychogenic	In hysteria and malingering
Therapeutic	Induced by electricity and excessive doses of insulin or metrazol
Poisonings	Strychnine picrotoxin and caffeine poisoning Alcoholism and plumbism
Cardiovascular	With neurovascular disturbances such as hemorrhage thrombosis and edema With angiospasms and urticaria particularly in the allergies (p 547) Complete heart block hypertensive toxemia of pregnancy and malignant hypertension. Erythroblastosis foetalis

vestigation is inaugurated in order to prevent further recurrence The immediate program may be tabulated as undernoted

1 —*If respiration is embarrassed* start artificial respiration and send for pulmotor

of the month give *dilantin* in doses of 0 1 gm (1½ grains) three times daily continuing the phenobarbital for the first few dilantin days

Abortive Treatment—Abortive treatment is rarely possible since there is but a brief interval between aura and convulsion The potentially useful measures of therapy include inhalations of *100 per cent oxygen* the ingestion of *sugar* intravenous injection of *sodium phenobarbital* the rectal administration of a suspension of starch paste containing *1 to 2 drams* of *paraldehyde* the use of an enema or a saline purge inhalations of *amyl nitrite* and relaxation in a warm bath

Treatment of the Convulsion—The physician rarely sees a convulsion the attack of petit mal or the psychic equivalents His main task in the presence of any of these is to prevent trauma Most epileptics are badly damaged as the result of overzealous therapy Teeth are loosened or knocked out by clumsy attempts to introduce a mouth gag and the throat is often traumatized during the same procedure

The patient is assisted gently to a recumbent position and protected from bruises during the course of the convulsion A gag is introduced if possible and bystanders are assured that recovery is imminent if only nature can be given an opportunity

Status Epilepticus—The treatment of status epilepticus requires heroic measures Intravenous injections of sodium phenobarbital or pentobarbital are given Lumbar puncture is performed to reduce increased intracranial pressure An enema is given and inhalations of oxygen containing *5 to 10 per cent* carbon dioxide are advised

Surgery—The surgery of epilepsy holds considerable promise in isolated instances If there are any localizing phenomena an *exploratory craniotomy* is performed and attempts are made to relieve the effects of presenting pathology such as pressure from a cranial exostosis the presence of meningeal adhesions or a neoplasm

Institutionalization—Epileptics with mental or moral deterioration frequent attacks and episodes of status epilepticus are best institutionalized particularly if the members of the family are unable to care for the sufferer In well conducted farms or colonies the epileptic receives the benefit of well regulated hygiene and is removed from the tensions of the large world in which he is distinctly handicapped

Epilepsy and Marriage—The offspring of the epileptic has increased hazard of inheriting the parental affliction Marriage is associated with a real risk of transmission with which both contracting parties should be thoroughly acquainted before the marriage is consummated In the majority of the instances the advice of the physician is not sought until the wedding has taken place Under these circumstances it seems advisable to urge the use of *contraceptive measures* (p 2502) unless there are formidable religious objections

THE GENERAL MANAGEMENT OF CONVULSIONS

The convulsive state is marked by involuntary contractions of skeletal muscle These may vary from barely perceptible localized twitches to massive disturbances involving all of the muscle mass Convulsions that

SECTION XI

THE EYE

Associate Editor FREDERICK H THEODORE M D

2—*If tongue is caught between teeth* insert a metal gag to prevent further injury

3—*Avoid hypodermic injection of drugs* until the cause of the condition can be ascertained If necessary administer rapidly acting barbiturates (sodium pentothal) as in generalized intravenous anesthesia (p 3923) Avoid opiates and alleged circulatory stimulants

4—*Obtain history of previous attacks* (epilepsy) or the *use of drugs* or hypodermic substances administered with therapeutic purpose or for criminal or suicidal intent

5—*Obtain urine*, by catheter if necessary, and *test* for albumin and sugar If glycosuric and acidotic administer insulin and dextrose intravenously

6—With hyperpyrexia or insolation give *sponge baths* to reduce fever (p 3785)

7—*In suspected poisoning* perform gastric lavage and later a colon irrigation

8—With *excessive hypertension* consider phlebotomy

9—With history of *trauma* get skull x rays and summon neurosurgeon

10—With *bloody spinal fluid* adopt principle of 'skillful neglect' unless there is a clear history of trauma

11—With *turbid spinal fluid* and *suspected infection* inaugurate antibiotic therapy with intravenous penicillin

12—With suspected *cerebral edema* give intravenous injection of 50 per cent sucrose or dextrose (p 3824)

13—In *Jacksonian convulsions* remove patient to hospital for complete neurologic survey and exploratory craniotomy if necessary

14—In the *interval between attacks* get x rays of skull, complete neurologic and psychiatric status examinations tests of spinal fluid electroencephalograms and fundus examinations

15—If *status epilepticus* is present try warm baths or complete chloroform anesthesia

CHAPTER 75

INTRODUCTION PHYSIOLOGY METHODS OF EXAMINATION AND TREATMENT

The practitioner is concerned with clinical ophthalmology for its importance in problems relative to the visual apparatus and its *diagnostic significance in the recognition of extra ocular phenomena* The sclera shows earliest evidences of jaundice pupillary abnormalities are among the most reliable manifestations of cerebrospinal syphilis the optic nerve reflects the changes of many widespread disturbances of the central nervous system and the retinal arteries and veins *which alone of the vascular struc*

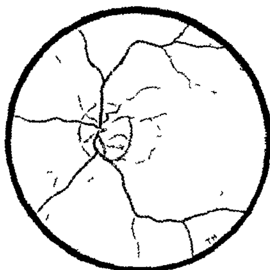


Fig 283 —Normal fundus with physiologic cupping

tures can be visualized often exhibit the herald signs of circulatory and renal disturbances at a time when prophylactic therapy is of immeasurable value

While it is not intended that the practitioner usurp the functions of the trained ophthalmologist (p 1540) he deprives himself and his patient of a vast fund of information unless he includes in his routine physical examination a careful survey of the eye particularly the information so readily available by the use of the *ophthalmoscope* (p 1545)

REVIEW OF ANATOMY

See p 3612

Gifford Textbook of Ophthalmology

Lpon entering the eye arterial pressure approximates 75 per cent f t h l l r nou pressure is slightly in excess of intra-ocular pressure Ma nt nan f tl

DIFFERENTIAL DIAGNOSIS OF

Tearing (Epiphora)

The phenomenon of tearing may be due to exces ive secretion or mechanical r er with lacrimal drainage The latte is always of local origin but profuse lacrimation a result from distant causation.

	DIAGNOSTIC FEATURES
Psychogenic	Emotional disturbances
Visual	Eye strain and asthenopia Order re act r (p 153.)
Lacrimal	Stenosis of na olacrimal duct E ersion o occlusion of lacrimal punctum Acute and chronic dacryocystiti Test patency by irri gation or probe (p 1557)
Adnexal	Ectropion, blepharochalasis (senile bagginess) or blepharitis Exophth lmos with lagophthal mos Note B.M.R.
Conjunctival	Foreign body photo retinitis burns and chem ical irritants or conjunctivitis Examine by oblique illumination (p 3622) Make smears and cultures of exudate and epithelial scrap-ings (p 1546)
Corneal and Scleral	Episcleritis scleritis and keratitis Refer to specialist for slit lamp examination and epi thelial scrapings
Uveal	Iridocyclitis with circumcorneal injection Refer to specialist for slit lamp examination Sympathetic ophthalmia following injury to sensitizing eye Urgent consultation with ophthalmologist
Disturbances of Intra-ocular Tension	Glaucoma with severe pain and increased tension Refer to specialist af er instillation of miotic for symptomatic relief (p 1548) Avoid examination with mydriatics (p 1548)
Neurogenic	With facial paralysis including Bell s palsy
Systemic Infections	Rhinitis with upper respiratory symptoms Measles with cutaneous rash Pertus is with paroxysmal cough and lymphocytosis Influ enza with respiratory manifestations and myalgias Mikulicz s disease with simultaneous swelling of submaxillaries
Allergy	Vasomotor rhinitis with nasal discharge con taining eosinophils Vernal conjunctivitis with eosinophils in local spread
Metabolic Disturbances and Poisonings	Hyperthyroidism with elevation of B M R and therapeutic response to iodine Senility alco-holism, exposure to tear-gas and paraphenyl endiamine poisoning

sh pa is of importance in the circulation of *intra-ocula fluid* and the pathogenesis of *glaucoma* (p 1578)

PHYSIOLOGY

The essential visual function of the ocular structures is dependent upon protective vascular motor and sensory components

PROTECTIVE MECHANISMS

Besides the protection of the bony orbit the eye is defended by fibrous coats movements of the lids lubricatory secretions and the sensitivity of the cornea

Lid Movements—Voluntary and involuntary movements of the lids are encountered In voluntary *blinking* occurs every two to ten seconds to moisten the cornea and sweep it clean Blinking helps drainage of tears and encourages the circulation of intra-ocular fluid See *Blepharospasm* (p 1610)

Sensitivity of the Cornea—Sensitivity of the cornea results in reflex blinking and an increased flow of tears at the slightest irritation Sensory fibers of the cornea are derived from the first division of the trigeminus

Tearing (Epiphora)—Lubrication of the eye is accomplished by tears from the lacrimal gland and secretions from mucous glands Tears are slightly salty and alkaline their bacterial activity is due to the presence of a *lysozyme* After cleansing the eyeball tears pass down the lacrimal passages to the nose The nervous mechanism that is involved in lacrimation has not been clearly worked out but it is interesting to note that psychic weeping occurs only in man

TABLE 101—DIFFERENTIATION OF CONJUNCTIVAL AND CILIARY INJECTION OF THE EYE

	Conjunctival Injection	Ciliary Injection
Involved Vessels	Posterior conjunctival	Posterior ciliary
Color	Brick red	Lilac
Location	Most marked in fornix with fading toward cornea	Circumcorneal with fading toward fornix
Appearance	Network of coarse anastomosing vessels Altered by pressure on lower lid	Small straight vessels radiating from cornea Unaffected by pressure on the lower lid
Discharge	Mucopurulent or purulent	Tears
Involved Tissue	Conjunctiva	Cornea iris or ciliary body

THE CIRCULATION

The conjunctiva is supplied with blood vessels from posterior conjunctival and anterior ciliary arteries and veins The iris is nourished by long posterior ciliary branches of the ophthalmic artery and these with the central artery of the retina constitute the circulatory mechanism of the organs of vision The vessels of the eye are influenced by nervous and chemical controls Vasoconstrictor fibers are present but vasodilator structures have not been identified The presence of axone reflexes has been proved by the elimination of painful phenomena after retrobulbar anesthetization

The distinction between conjunctival and ciliary injection assists in the differential diagnosis of ocular disease See Table 101

Transparency of the refractory media (cornea aqueous lens vitreous and anterior layers of retina) requires a state of avascularity hence the nutrition of these structures is maintained by a process of diffusion The refracting media are highly susceptible to metabolic disturbances are infrequently attacked in systemic infection and possess little resistance once invasion has occurred The *uveal tract* (p 3619) by contrast is the vascular portion of the eye it suffers from few metabolic diseases but infection occurs with greater frequency and at immense threat to visual functions

Upon entering the eye arterial pressure approximates 7 per cent of brachial readings enous pressure is slightly in excess of intra-ocular pressure Maintenance of these relat on

DIFFERENTIAL DIAGNOSIS OF

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The phenomenon of tearing may be due to excessive secretion o mechanical interference *with lacrimal drainage The latter is always of local origin but profuse lacrimation also may* result from distant causation

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Visual	Eye strain and asthenopia *Order refraction* (p 153)
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h ps is of importance in the circulati n of *intra-ocula fluid* and the pathog nes s of *glaucoma* (p 1578)

INTRA OCULAR FLUID

Intra ocular fluid is of complex chemical composition and contains elements present in blood serum It appears to be a *dialysate* of capillary blood but may be a secretion of ciliary epithelium

Circulation of Intra Ocular Fluid—The circulation of intra-ocular fluid does not follow a definite pathway A constant renewal probably occurs through the vascularized tissues of the eye As a result of external pressure and thermal stimuli there is an intermittent flow from ciliary bodies through pupils into anterior chambers and out through the canals of Schlemm

Intra Ocular Pressure—Normal intra-ocular pressure (p 1578) measures between 12 and 30 mm of mercury Tension varies 1 to 2 mm with the pulse beat, and rises 3 mm on inspiration It is highest in the early morning drops sharply on arising and increases again at night to reach its maximum The normal diurnal variation is 2 to 3 mm of mercury but in abnormal conditions such as *glaucoma* the swings are wider

Intra-ocular pressure is maintained by the elasticity of the outer coat of the eye and the volume of intra ocular fluid A safety valve action of the canal of Schlemm maintains relatively constant conditions

Fig 285—Scheme of movements of the cornea of the right eye from the resting position with the muscles that affect them The predominant muscles are in capital letters the roman numerals after the names indicate the nerve supply (Adapted from Testut 1914)

Variations in Intra Ocular Pressure—Intra ocular pressure is subject to variations due to physiological and pathological conditions *Pressure on the globe* and the actions of extra ocular muscles raise the tension 4 to 10 mm of mercury Alterations in *arterial blood pressure* rarely causes corresponding changes in intra-ocular pressure but *venous pressure* changes are delicately reflected Occlusion of the retinal vein for example causes late glaucoma

Pressure of intra-ocular fluid is influenced by alterations in the volume of the contents of the eye With a general increase in the osmotic pressure of blood the eye loses fluid and tension drops This principle is utilized in the treatment of acute glaucoma (p 1578)

OCULAR MOTILITY

The eye is rotated around three axes by extra-ocular muscles Each eye movement involves contraction of active muscles and inhibition of antagonists Rotation about the *vertical axis* is an *adduction* when the eye is turned toward the nose and an *abduction* when the eye moves toward the temple Around the transverse axis the eye is e

Torsion movements about the *anteroposterior axis* are less obvious when the upper end of the vertical meridian is tilted toward the nose it is an *intorsion* and when it is tilted toward *the temple or outward it is an extorsion.*

The Cardinal Directions of Gaze—The field of muscle action is that direction in which *its main action is greatest.* Ocular movements are *examined in six cardinal directions of gaze* Obvious pareses and overactions become grossly apparent, but the detection of slight involvement which is sufficient to *cause diplopia (p 1523) requires screen testing by the expert*

Conjugate Movements—Regulation of ocular movements to achieve simultaneous action *is accomplished in part by* various *of the cerebral association centers* In associated or

TABLE 102—OCULAR FUNCTIONS OF EXTRA-OCULAR MUSCLES

Muscle	Main Action	Subsidiary Action
Superior Rectus	Elevation Action increases as eye is turned out, becomes nil when eye is turned in	Adducts eye and rotates vertical meridian inward (*intorsion*) Action increases as eye is turned in
Inferior Oblique	Elevation Action increases as eye is turned in becomes nil when eye is turned out.	Abducts eye and rotates vertical meridian outward (extorsion) Action increases as eye is turned out
Inferior Rectus	Depression Action increases as eye is turned out, becomes nil as eye is turned in	Adduction and extorsion Action increases as eye is turned in
Superior Oblique	Depression Action increases as eye is turned in becomes nil as eye is turned out.	Abduction and intorsion Action increases as eye is turned out
Medial Rectus	Medial rotation	None
Lateral Rectus	Lateral rotation	None

TABLE 103—PREDOMINANT MUSCLE ACTIONS INVOLVED IN EYE MOVEMENTS

Eyes Directed To	Muscles Predominantly Acting
Right	R lateral rectus L medial rectus
Left	R medial rectus L lateral rectus
Up and right	R *superior rectus L inferior oblique*
Up and left	R inferior oblique L superior rectus
Down and right	R inferior rectus L superior oblique
Down and left	R superior oblique L inferior rectus

conjugate movements the visual axes may be parallel or disjunctive in the latter type the visual axes may converge (*convergence*) or diverge (*divergence*) Loss of one or more conjugate movements may occur in disease despite normality of extra-ocular musculature

Binocular Vision—The complicated mechanism of ocular motility is designed to insure *binocular fixation* by which the *image of an object falls* on the macula and corresponding points of the retina of each eye The two images are then fused into a single mental image in a theoretical cerebral *fusion center* This *faculty* in its highest form, permits stereoscopic or solid vision.

Failure of the image of an object to fall on *corresponding points of* the two retinas due

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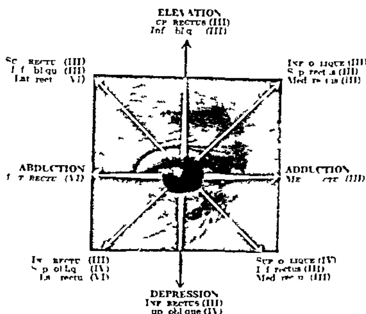


Fig 285 —Scheme of movements of the cornea of the right eye from the resting position with the muscles that affect them The predominant muscles are in capital letters the roman numerals after the names indicate the nerve supply (Adapted from Testut, 1914)

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Diplopia tests performed by the specialist to determine the muscle or muscles involved are accomplished by placing a red lens in front of one eye while moving a small light (ophthalmoscope bulb) in the six cardinal directions of gaze The examiner questions the patient concerning the amount of separation of red and untinted images Separation is greatest in the field of action of the involved muscle (Table 104) The true image corresponding to the fixing eye is distinct as it falls on the macula the "false" image of the deviating eye is less distinct.

Suppression —Suppression is a mental process by which the image of the deviating eye is not allowed to enter consciousness By this means unpleasant symptoms associated with double images are automatically eliminated In *concomitant strabismus (p 1529)* a defective fusion faculty is at fault and suppression is accomplished at the onset of the squint In paralytic strabismus of long standing the image of one eye usually the paralyzed eye may be entirely suppressed

OCULAR DEVIATIONS (PARALYTIC AND NONPARALYTIC STRABISMUS)

Ocular deviations may be paralytic (noncomitant) or nonparalytic (concomitant)

Paralytic Strabismus —The important characteristics of paralytic strabismus of the ocular muscles are (1) limitation of eye movement in the field of action of the paralyzed muscle,



A



B

Fig 296 —Right oculomotor paralysis *A* Primary position left eye fixing *B* Eyes left, right eye is held in abduction by contracture of external rectus

(2) increasing deviation of the affected eye as the eyes are turned into the field of action of the involved muscle (3) diplopia which becomes more marked in the field of action of the paralyzed muscle and (4) associated symptoms of nausea and vertigo

Nonparalytic Strabismus —Nonparalytic or concomitant strabismus (*heterotropia*) is the common type of *cross eye* encountered in childhood In contradistinction to the characteristics of paralytic strabismus (1) the deviation from parallelism does not vary but is always the same in amount in every direction of gaze (2) the power of the individual muscles is normal, *as is demonstrable by covering one eye and having the patient rotate the other eye in all directions (3) due to suppression neither diplopia nor other subjective symptoms are* encountered (4) the two eyes do not work as a pair the one sighting the envisioned object being the *fixing eye* while the other is the *squinting eye*

Squint.—Squint arises *from a disturbance of the delicate* balance between the powers of convergence and divergence in individuals with a congenital defect of the fusion faculty or the innate desire for binocular single vision In *monocular squint* the one eye constantly deviates and the other fixes vision in the *deviating eye* is generally poor In *alternating squint* the patient fixes with either eye and vision in each is good

Concomitant Strabismus (Heterotropia) —According to the direction of deviation *concomitant squints are convergent* (internal strabismus or esotropia) *divergent* (external

to deviation of one eye results in *diplopia* (*double vision*) (p 1598) Due to the process of *projection* by which images striking the *upper* portion of the retina (for example) are in

DIFFERENTIAL DIAGNOSIS OF

Double Vision (Diplopia)

In double vision there is the recording of a true and a false image The latter may be situated laterally above or below the observed object

	DIAGNOSTIC FEATURES
Paralytic Strabismus	Of third, fourth or sixth cranial nerves Limitation of eye movement only in field of action of paralyzed muscles (p 1527) Complete neurologic examination and check cerebrospinal fluid (p 3734)
Nonparalytic Strabismus	Heterotropia of childhood Power of individual muscles is normal Screen test (p 1543)
Errors of Refraction and Accommodation	Asthenopia (eyestrain) Order refraction (p 1537)
Ophthalmic	Dislocation of lens Orbital cellulitis or tumors Symblepharon Exophthalmos Supplement local inspection with reference to specialist and estimation of B M R
Otogenic	Mastoiditis petrositis and labyrinthitis Examine ear-drums (p 2142) Get x rays of mastoid region (p 2146) Refer to otologist.
Neurogenic	Neoplasm inflammation, trauma or vascular injury to cerebrum or cerebellum Get neurologic status and examine fundi (p 3629) Suppurative and non suppurative encephalitides and meningitides Examine cerebrospinal fluid and get blood for virus neutralizing bodies (p 59) Tabes dorsalis and general paresis with positive serology and characteristic gold curves of cerebrospinal fluid Multiple sclerosis with pallor of optic disk, tremor and disseminated neurologic manifestations
Myogenic	Myasthenia gravis with therapeutic response to neostigmine (p 2886)
Psychogenic and Functional	Neuroses and hysteria Fatigue Ophthalmoplegic migraine
Metabolic	Diabetes mellitus with glycosuria and hyperglycemia Hyperthyroidism with elevation of B M R. and therapeutic response to iodine (p 611) Vitamin deficiency with therapeutic response to thiamine and niacin (p 616)
Poisonings	Alcoholism Addiction to cocaine Over-dosage with barbiturate Exposure to paraphenylenediamine
Systemic Infection	Diphtheria with ulceromembranous exudate containing organisms Botulism with history of ingestion of contaminated canned food

terpreted as coming from the *lower half* of the visual field diplopia is opposite in character to ocular deviation

Diplopia tests performed by the specialist to determine the muscl or muscles involved are accomplished by placing a red lens in front of one eye while moving a small light (ophthalmoscope bulb) in the six cardinal directions of gaze The examiner questions the p tient concerning the amount of separation of red and untinted images Separation is greatest in the field of action of the involved muscle (Table 102) The true image corre ponding to the fixing eye is d stinct as it falls on the macula the false image of the deviating eye is less d stinct.

Suppress on—Suppression is a mental process by which the image of the deviating eye is not allowed to enter consciousness By this means unpleasant symptoms associated with double images are automatically eliminated In concomitant strab ismus (p 1529) a defective fusion faculty is at fault and suppression is accomplished at the onset of the squint In paralytic strabismus of long standing th image of one eye u ually the paral zed eye may be entirely suppressed

OCULAR DEVIATIONS (PARALYTIC AND NONPARALYTIC STRABISMUS)

Ocular deviations may be paralytic (noncomitant) or nonparalytic (concomitant)

Paralyt c St ab smus—The important characteristics of paralytic strabismus of the ocular muscles are (1) limitation of eye movement in the field of action of the paralyzed muscle



A B

Fig 286—Right oculomotor paralys s A Primary position left eye fixing B Eyes left right eye is held n abduction by contracture of external rectus

(2) increas ng deviation of the affect d eye as the eyes are turned into the field of action of the involved muscle (3) diplop a which becomes more marked in the field of action of the paralyzed muscle and (4) associated symptoms of nausea and vertigo

Nonparalyt c Strab mus—Nonparalytic or concomitant strabismus (*heterotropia*) is the common type of *cross eye* encountered in childhood In contradist nct on to the characteris tics of paralytic strabismus (1) the dev ation from parallelism does not vary but is always the same in amount in every d rection of ga e (2) the power of the individual muscles is normal as is demonstrabl by covering one eye and having the patient rotate the other eye in all d rections (3) due to suppression neither d plop a nor other subjective symptoms are encountered (4) the two eyes do not work as a pair th one sighting the envis oned object be ng the *fixing eye* wh l the other is the *squ nting* ye

Squint—Squ nt arises from a d sturbance of the delicate balance between the powers of convergence and divergence in individuals with a congenital defect of the fusion faculty or the innate desire for binocular single vision In *monocular squint* the one eye constantly deviates and the other fixes vision in the dev ating eye is generally poor In *alternating squ nt* the patient fix s w th e ther eye and vision in each is good

Concom tant Strab smus (H te otrop)—According to the direction of deviation con com tant squints are *convergent* (nternal) strabismus or esotrop a) *dive gent* (external

to deviation of one eye results in *diplopia (double vision)* (p 1528) Due to the process of *projection* by which images striking the *upper* portion of the retina (for example) are in

DIFFERENTIAL DIAGNOSIS OF

Double Vision (Diplopia)

In double vision there is the recording of a true and a false image The latter may be situated laterally above or below the observed object

	DIAGNOSTIC FEATURES
Paralytic Strabismus	Of third fourth or sixth cranial nerves Limitation of eye movement only in field of action of paralyzed muscles (p 1527) Complete neurologic examination and check cerebrospinal fluid (p 3734)
Nonparalytic Strabismus	Heterotropia of childhood Power of individual muscles is normal Screen test (p 1543)
Errors of Refraction and Accommodation	Asthenopia (eyestrain) Order refraction (p 1537)
Ophthalmic	Dislocation of lens Orbital cellulitis or tumors Symblepharon Exophthalmos Supplement local inspection with reference to specialist and estimation of B M R.
Otogenic	Mastoiditis petrositis and labyrinthitis Examine ear-drums (p 2142) Get x rays of mastoid region (p 2146) Refer to otologist
Neurogenic	Neoplasm, inflammation, trauma or vascular injury to cerebrum or cerebellum. Get neurologic status and examine fundi (p 3629) Suppurative and non suppurative encephalitides and meningitides Examine cerebro spinal fluid and get blood for virus neutralizing bodies (p 59) Tabes dorsalis and general paresis with positive serology and characteristic gold curves of cerebrospinal fluid. Multiple sclerosis with pallor of optic disk tremor and disseminated neurologic manifestations
Myogenic	Myasthenia gravis with therapeutic response to neostigmine (p 2886)
Psychogenic and Functional	Neuroses and hysteria Fatigue Ophthalmoplegic migraine
Metabolic	Diabetes mellitus with glycosuria and hyperglycemia Hyperthyroidism with elevation of B M.R. and therapeutic response to iodine (p 611) Vitamin deficiency with therapeutic response to thiamine and niacin (p 616)
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Systemic Infection	Diphtheria with ulceromembranous exudate containing organisms Botulism with history of ingestion of contaminated canned food

terpreted as coming from the *lower half* of the visual field diplopia is opposite in character to ocular deviation

adrenergic or sympathetic fibers (p 1390) from the antagonistic subdivision of the involuntary nervous system

The pupilloconstrictor center is probably located in the accessory third nerve nucleus of Edinger Westphal Efferent fibers follow the third cranial nerve to the ciliary ganglion from which they pass to the eye by way of short ciliary nerves The pupillodilator center is the ciliospinal area of Budge Between pupilloconstrictor and pupillodilator centers there is a partial decussation and preganglionic fibers travel through the cervical sympathetic to the superior cervical ganglion whence post ganglionic fibers enter the skull with the carotid plexus, travel over the gasserian ganglion and accompany the ophthalmic division of the fifth cranial nerve to join the long ciliary nerves as they enter the eyeball In this way they avoid the ciliary ganglion

Innervation of Intra Ocular Muscles —The *dilator* muscle of the iris is innervated solely by adrenergic fibers Instillation of sympathomimetic amines such as epinephrine produces active contraction of the muscle and resultant mydriasis Paralysis of the nerve mechanism through destruction of the superior cervical ganglion (Horner's syndrome) results in miosis with narrowing of the palpebral fissure and enophthalmos

TABLE 104 —THE VARIETIES OF STRABISMUS

	Paralytic Strabismus	Heterotropia	Heterophoria
Limitation of eye movement	In field of action of paralyzed muscle	None	None
Effect of turning eye into field of action of involved muscle	Increased deviation	*No increase in deviation from parallelism*	*No increase in deviation from parallelism*
Diplopia	More marked in field of action of involved muscle	None	Equal in all fields
Associated symptoms	Nausea Vertigo	Non	Nausea, Vertigo
Screen test	When squinting eye is covered and uncovered no movement occurs in either eye When fixing eye is covered and uncovered both eyes move	When squinting eye is covered and uncovered no movement occurs in either eye When fixing eye is covered and uncovered both eyes move	Either eye when covered deviates but tends to swing back into place on uncovering

The *sphincter* muscle has double innervation being provided with stimulatory cholinergic nerves and inhibitory adrenergic nerves Depression or paralysis of the cholinergic mechanism by atropine and drugs of similar action produces mydriasis while stimulants such as physostigmine result in miosis

Pharmacology of Pupillary Reactions —Cholinergic nerve endings act through the liberation of acetylcholine which initiates the action of the innervated structure Acetylcholine is rapidly destroyed in the body by a choline esterase which is present in all tissues Pharmacologically the eff ct of *acetylcholine may be simulated by the instillation of available* derivatives such as acetyl beta methylcholine (mecholyl) and carbaminoylcholine (doryl) The latter exerts more powerful miotic effect since it is inactivated by tissue esterase only after several hours

Rapid destruction of choline compounds is inhibited by drugs with a carbamate group such as physostigmine (eserine) and neostigmine (p 1391) These combine and inactivate choline esterase and cause paralysis of the dilator mechanism with the production of *miosis* Pilocarpine and muscarine produce a similar effect but their mechanism of action is dissimilar since they are not destroyed by cholinesterase

...nic nerve endings are stimulated by liberation of an epinephrine-like substance

strabismus or exotropia) or *vertical* (hypertropia) *Accommodative strabismus* exists when an uncorrected error of refraction causes excessive accommodation and excessive convergence *Non-accommodative strabismus* usually arises as a result of lack of use of an eye due to *anisometropia* (p 1536) which is a marked refractive difference between the two eyes or to impaired vision in an eye due to disease In the latter instance the blind eye generally turns out (exotropia)

Latent Strabismus (Heterophoria).—Latent strabismus (heterophoria) is the condition in which the eyes have a tendency to deviate but, due to a well functioning fusion faculty maintain binocular single vision with effort As the deviation is latent and not apparent, it must be demonstrated by the screen test in which fusion is broken According to the deviating tendency phorias are classified as *esophoria exophoria hyperphoria* and *hypophoria* A tendency of the vertical meridian to deviate from the vertical position is *cyclophoria*

Clinical Manifestations of Strabismus—Heterotropia gives no symptoms other than those resulting from the cosmetic appearance heterophorias may produce asthenopia headache blurring of vision diplopia, nausea and vertigo

Treatment of Strabismus—Concomitant strabismus (heterotropia) is treated by the specialist by the use of glasses orthoptic training and surgery or a combination of all three In accommodative squint the use of proper glasses may diminish the strabismus considerably or even cure it Orthoptic training or exercises of considerable value in some cases are



Fig 287—*A* Concomitant convergent strabismus *B* Result of recession and advancement

attempts to establish the simultaneous use of both eyes It is often necessary to resort to *occlusion of the good eye* by a patch *or to diminution of the function of the good eye* by the prolonged use of atropine in order to stimulate vision in the squinting or amblyopic eye This necessary preliminary to orthoptic training should not be attempted if the vision of the eye cannot be improved to 20/70 The best results of orthoptic training are obtained in children between the ages of three to six years with exotropia or the phorias especially convergence insufficiency

After a trial of non-operative procedures for at least a year operation is indicated Surgery may be performed at any age and early operation has definite psychological advantages for the pre-school child Postoperative orthoptic training often is much more valuable than preoperative efforts

The treatment of *heterophoria* consists of correction of any associated refractive error by means of glasses the incorporation of prisms in hyperphoria and of orthoptic training Surgery is utilized rarely as a last resort

INTRA OCULAR MUSCLES AND PUPILLARY REACTIONS

Beside voluntary extra-ocular muscles the eye possesses unstriated muscles which control pupillary reactions The *sphincter of the pupil and its ciliary muscle are innervated by the* vagal or cholinergic system (p 1588) through oculomotor nerves The *dilator* is supplied by

* Gifford, Textbook of Ophthalmology

adrenergic or sympathetic fibers (p 1390) from the antagonistic subdivision of the involuntary nervous system

The pupilloconstrictor center is probably located in the accessory third nerve nucleus of Edinger Westphal Efferent fibers follow the third cranial nerve to the ciliary ganglion from which they pass to the eye by way of short ciliary nerves The pupillodilator center is the ciliospinal area of Budge Between pupilloconstrictor and pupillodilator centers there is a partial decussation and preganglionic fibers travel through the cervical sympathetic to the superior cervical ganglion whence post ganglionic fibers enter the skull with the carotid plexus travel over the gasserian ganglion and accompany the ophthalmic division of the fifth cranial nerve to join the long ciliary nerves as they enter the eyeball In this way they avoid the ciliary ganglion

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nerve endings are stimulated by liberation of an epinephrin like substance

This effect is produced pharmacologically by cocaine ephedrine neosynephrine benzedrine and paredrine

Physiology of the Pupil—The pupil acts as a diaphragm for the eye It regulates the amount of light that falls on the retina contracting when there is high intensity and dilating when the intensity of illumination is low In this manner it improves the effectiveness of the eye as an optical instrument

The normal diameter of the pupil is about 3.5 mm It is wider in those with myopia and in the female It is smaller in those with hyperopia and at the extremes of age A pupil which is smaller than 2 mm under normal illumination is abnormally *miotic* (p 1533) One that is larger than 5 mm under similar conditions is normally *mydriatic* (p 1533) Inequality in the size of the two pupils is an *anisocoria* (p 1534) which may be physiologic or pathologic

Pupillary Reflexes—The normal pupil responds to a variety of sensory stimuli many of which have important significance in clinical medicine

Direct and Indirect (Consensual) Light Reflexes—Exposure of the retina to bright light results in contraction of the pupil (*direct light reflex*) The degree of effect varies with the relative change of illumination the state of adaptation of the eye and the intensity of the light stimulus The pupil of the unstimulated eye under normal circumstances should contract simultaneously and to equal degree (*indirect or consensual light reflex*)

The Near Reflex (Accommodation)—Focus of the eye on a near object results in accommodation convergence and pupilloconstriction The miosis occurs normally when accommodation and convergence are eliminated respectively by lenses and prisms The independence of light and accommodation reflexes is illustrated by the Argyll Robertson pupil (p 1534)

The Orbicularis or Lid Reflex—With the eye closed the pupil of the shut eye contracts as a reflection of a central mechanism

The Oculosensory or Trigeminal Reflex—The application of sensory stimuli other than light to the eye and its adnexa results in initial pupillodilatation followed by contraction The response is bilateral and is a true reflex mediated through the trigeminal nerve

Psychosensory Reflex—Stimulation of any sensory nerves except for those supplying the eye and its adnexa results in pupillary dilatation through a central response in the cortex

THE PHYSIOLOGY OF VISION

When light strikes the retina there occur chemical and electrical changes *Physical changes* include phototropic migration of retinal pigment toward the light contraction of the cones and swelling of the rods *Chemically* retinal tissue becomes acid and visual purple or rhodopsin is bleached a change in which *vitamin A* participates (p 617) The chemical reaction is reversible and is associated with measurable *electrical potential*

Scotopic and Photopic Vision (Duplicity Theory)—According to the duplicity theory two types of visual activity exist in the retina By means of the rods low intensities of light are perceived the scotopic type of vision is achromatic and is concerned with the appreciation of light and movement it is especially evidenced in conditions of dark adaptation Higher intensities of light are registered by the cones (*photopic vision*) which concern themselves with form and color vision Photopic vision is particularly evident in the light adapted eye with its relatively high threshold stimulus intensity

The Visual Sensations—Stimulation of the retina results in three types of visual sensation related to light, color and form

Light—Light sense is the faculty by which the eye distinguishes between light and dark and between differences in the brightness of illuminated objects This faculty is more sensitive than color or form senses and operates when illumination is sufficiently diminished to eliminate other types of sensations In the dark adapted eye an extremely slight degree of brightness evokes a sensation of light Disturbances of light sense (p 1535) consist of *night blindness* and *day blindness*

Color—Appreciation of color is a function of the cones or of the photopic mechanism of vision In good illumination the upper end of the spectrum appears brighter especially yellow In poor illumination the lower end of the spectrum especially blue is most easily visible (Purkinje s phenomenon) The *Young Helmholtz theory* of color vision postulates that all hues can be compounded from a mixture of the three primary spectral colors of red, green and violet. Normal color vision according to this theory is *trichromatic* and the retina contains three types of cones capable of producing sensations of red green and violet. Anomalies of color sense (p 1535) consist of *color blindness* and *perversions of col*

DIFFERENTIAL DIAGNOSIS OF

Abnormalities of Pupillary Diameter

The pupils may be excessively dilated (mydriasis) or contracted (miosis) Differences in pupillary size constitute anisocoria which is indicative of the unilateral operation of any of the provocative factors which cause bilateral mydriasis or miosis

	MYDRIASIS	MIOSIS
Congenital	Neuromuscular with normal vision Amaurotic with optic nerve atrophy as in amaurotic family idiocy	Neuromuscular with normal vision
Refractive	Myopia (p 1536)	Hyperopia (p 1536)
Oculomotor	Paralyses with associated ophthalmoplegia (p 1537)	Stimulation usually reflex and spastic
Disturbances of Superior Cervical Sympathetic	Irritation due to hyperthyroidism upper lobe pneumonia or unilateral pleurisy Get B M R in afebrile and chest film in the presence of fever	Paralysis from pressure of aortic aneurysm, mediastinal neoplasm or lymphadenopathy Get chest film Injury or surgical destruction of ganglion causing Horner s syndrome with enophthalmos and narrowing of palpebral fissure
Ophthalmic	Glaucoma with increased intraocular tension Keratitis with observable lesion Amaurosis due to optic nerve atrophy degenerative retinitis or occlusion of retinal arteries Note ophthalmoscopic findings (p 3628)	Keratitis and iritis with circumcorneal injection (p 1524) Slit lamp examination (p 1544)
Neurogenic	Paralysis of pupilloconstrictor or stimulation of pupillodilator centers from cerebral tumor vascular accident or encephalitis Note other neurologic changes Tabes dorsalis general paresis and syphilitis meningitis with positive serology and changes in cerebrospinal fluid. Suppurative and non suppurative encephalitides and meningitides with other neurologic findings and changes in cerebrospinal fluid. Multiple sclerosis syringomyelia and transverse myelitis with other motor and sensory findings	Stimulation of pupilloconstrictor or paralysis of pupillodilator mechanisms from same causes as produced mydrias s
Pharmacodynamic	Atropine and atropine like substances Sympathomimetic amines such as epinephrine and related substances Acute alcoholism and overdosage with general anesthetics hypnotics and sedatives	Physostigmine and allied substances Opiate and morphine poisoning

DIFFERENTIAL DIAGNOSIS OF

Pupillary Abnormalities Other Than Mydriasis and Miosis

Other than dilatation and contraction abnormalities of the pupil may have important diag nostic significance mo t particularly but not exclusively in cerebrospinal syphilis

	DIAGNOSTIC FEATURES
Irregular Pupils	Glaucoma with increased intraocular tension *Iritis and keratitis with changes observed in* slit lamp and circumcorneal injection (p 1534) General paresis tabes dorsalis and syphilitic meningitis with positive serology and characteristic changes of the gold curv (p 3736)
Argyll Robertson Pupil (Paralysis of Light Re flex with Retention of Near Reflex or Accom modation)	Glaucoma with increased intraocular tension General paresis tabes dorsalis and syphilitic meningitis Organic neurologic conditions, such as encephalitis meningitis poliomyelitis *multiple neuritis due to diabetes* mellitus or avitaminosis multiple sclerosis and syringo myelia Complete neurologic examination and tests of blood urine and spinal fluid
Inverse Argyll Robertson Pupil (Paralysis of Near Reflex with Retention of Light Reflex)	Cerebrospinal syphilis with positive findings in blood and spinal fluid Diphtheria with ulceromembranous inflammation Tumors of corpora quadrigemina with other neurolog c findings
Paralysis of Light and Near Reflex but Reten tion of Lid Reflex	Supranuclear lesions at the termination of afferent paths Refer to specialist consultant.
Loss of Psychosensory Reflex	Severe organic disturbances such as idiocy imbecility chronic alcoholism and general paresis Examine blood and spinal fluid
Hippus (Rhythmic Contractions and Dilatation of Pupils)	With paralysis of internal and external ocular muscles in hemiplegia multiple sclerosis and meningitis Get complete neurologic survey and check cerebrospinal fluid (p 3734)
Pupillary Nystagmus	With ocular nystagmus in epidemic enceph alitis and hypertensive toxemia of pregnancy
Cyclic Oculomotor Paralysis (With alternating Phases of Elevation of Upper Lid, Contraction of Pupil Convergence and Accommodation followed by Ptosis Mydriasis and Pupillo-paralysis)	Congenital anomaly
Springing Pupil (Sudden momentary Pupillo-dilatation)	Cerebrospinal syphilis with positive findings in blood and spinal fluid (p 3736) Neuras thenia Veronal poisoning (p 3836)
Myotonic Pupillary Reaction (Sluggish Light Reflex with Continuation of Miosis after Re moval of Stimulus)	With absent knee jerks in Ad e s syndrome a non syphilitic affliction Note normal blood and spinal fluid

Form—Form sense is concerned with the abil ty of the eye to perceive the shape of ob jects In practice form sense is measured by charts of *visual acuity* (Snellen) It is a photopic mechanism l pendent upon the retina cones of which the human eye possesses 200 000 to the square millimeter The unusually keen vision of the eagl is said to be du to its cone population at the fovea, of 1 000 000 cones to the square millim ter

THE EYE AS AN OPTICAL INSTRUMENT

In passing through the eyeball to be converged on the retina rays of light are refra ted by cornea and anterior and posterior surfaces of the crystalline lens

DIFFERENTIAL DIAGNOSIS OF

Disturbances of Color, Light and Form Sense

Disturbances of the color light and form senses are not often encountered, though they may be of important diagnostic significance

	DIAGNOSTIC FEATURES
Color Blindness (Achromatopsia)	Congenital anomaly Hysteria Malingering Optic atrophy or neuritis Examine fundus and test with pseudo-isochromatic charts (p 1541)
Perversions of Color Sense (Chromatopsia)	Green vision with digitalis Yellow vision with picric acid and santonin Erythropsia (red vision) following cataract extraction Xan thopsia (yellow vision) in jaundice
Day Blindness (Hemeralopia)	Albinism, Toxic amblyopia colobomas and irideremia Refer to specialist
Night Blindness (Nyctalopia)	Vitamin A deficiency with response to thera peutic tests Oguchi s disease in Japanese Simple glaucoma with elevation of intra-ocular tension Choroideremia and retinitis pigmen tosa—refer to specialist Hysteria
Metamorphopsia (Changes in the Shape of Ob jects)	
Micropsia and Macropsia (Changes in the Size of Objects)	Exudative choroiditis with circumcorneal in jection and ophthalmoscopic findings (p 1534) Retinitis and detachment of the retina with fundus changes Refer to specialist
Photopsia (Flashes of Light and Bright Circles)	Migraine and epilepsy with characteristic history Exudative choroiditis and detachment of retina with fu ndus changes

R f act on —Rays of light pass out in all directions from any lum nous po nt Rays which emanate from a source less than 20 feet distant diverge but rays travell ng in excess of 20 feet are assumed to be parallel

When the ray of light meets an opaque body it is *absorbed* or *reflected* A transparent body permits pas age of the greater part of the ray with some deflection Bending of the ray is described as *ef act on* The *ndex of ref action* is the comparative length of time that it takes light to t avel a defin t d stance in d fferent transparent med a If a r is taken as unity the index of refraction of water is 1 33 of the cornea 1.33 of the l ns 1 40 and of crown glass of the type usually used in optical procedures 1.5

Acc mmodat on—The ability or power to increase the refract ve or focus ng power of the eye is *accommodat on* Th s is accomplished by an increase in convexity of the crystalline lens According to the Helmholtz theory the ci ary muscle contracts the choroid is drawn forward to r lax th suspensory l gament or zonul tens on on th l ns capsul is released

DIFFERENTIAL DIAGNOSIS OF

Pupillary Abnormalities Other Than Mydriasis and Miosis

Other than dilatation and contraction abnormalities of the pupil may have important diag nostic significance most particularly but not exclusively in cerebrospinal syphilis

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Irregular Pupils	Glaucoma with increased intraocular tension Iritis and keratitis with changes observed in slit lamp and circumcorneal injection (p 1534) General paresis tabes dorsalis and syphilitic meningitis with positive erology and characteristic changes of the gold curve (p 3736)
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Loss of Psychosensory Reflex	Severe organic disturbances such as idiocy imbecility chronic alcoholism and general paresis Examine blood and spinal fluid
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Pupillary Nystagmus	With ocular nystagmus in epidemic encephalitis and hypertensive toxemia of pregnancy
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Myotonic Pupillary Reaction (Sluggish Light *Reflex* with Continuation of Miosis after Re moval of Stimulus)	With absent knee jerks in Adie s syndrome a non syphilitic affliction Note normal blood and spinal fluid

in front of the retina *Astigmatism* is a more complicated disturbance in which there is an abnormality of refraction which differs in the several meridians

DISTURBANCES OF ACCOMMODATION

The principal disturbance of accommodation is *presbyopia* in which advancing age results in a physiological weakness of the accommodation mechanism and the characteristic inability of the older individual to focus for near vision *Spasm of accommodation* occurs more frequently in younger patients and is in all likelihood a neuromuscular disturbance often of toxic origin In *anisometropia* there is inequality in refraction with consequent visual difficulty With *aniseikonia* the retinal images are unequal

ASTHENOPIA (EYESTRAIN)

Eyestrain may result in a variety of symptoms The purely visual complaints include blurring of vision lacrimation blinking blepharospasm spots before the eyes a sandy feeling in the eyes and redness of the conjunctiva The more distant complaints include headache vertigo and nausea

Treatment—Asthenopia requires both systemic and local therapy The patient with eyestrain should be given instructions as to correct illumination the avoidance of eyestrain and the means of preventing physical fatigue Weakness of accommodation may be ameliorated by eye exercises spasms are sometimes alleviated by discontinuance of smoking and the use of mild sedatives such as sodium phenobarbital 15 mg (¼ grain) with or without an antispasmodic such as pavatrine 2 mg (1/30th grain) Main reliance however in the ametropias and presbyopia is placed upon the correctly fitted eyeglass

Eyeglasses—The refractive properties of glass are employed in the prescription of corrective spectacles which contain prisms or lenses Hyperopia and presbyopia are compensated by convex spherical lenses while myopia is improved by concave spherical lenses An eyeglass prescription for astigmatism is considerably more complicated and is specialist province

Prisms—A prism is a piece of glass or other substance bounded by plane surfaces inclined toward each other The thin edge where the surfaces meet is the *apex* and the opposite thick portion is the *base* Rays of light passing through a prism are bent toward the base Since the eye sees things reversed an object seen through a prism *appears* displaced toward the apex Prisms are used (*1*) to counteract the effects of ocular deviations due to muscle pareses (*2*) to exercise weak muscles (*3*) to test muscular power (*4*) to measure and test for heterophoria and heterotropia (p 1531)

Lenses—A lens is a transparent refracting medium usually glass in which one or both surfaces are curved There may be spherical or cylindrical lenses *Spherical lenses* are segments of spheres which refract rays of light equally in all meridians or planes *Convex spherical* or *plus* (+) *lenses* are thick at the center and thin at their edges They may be considered as two prisms with bases joined together in the center they converge parallel rays to a focus *Concave spherical lenses* or *minus* (—) *lenses* are thin in the center and thick at the edges They may be considered a

and the elastic capsule moulds the softer cortex into a more spherical shape Associated with accommodation contraction of the pupils and convergence occur

When the normal or emmetropic eye is at rest parallel rays from a distant object are focused on the retina but rays coming from a near object are focused behind the retina and that object appears blurred By accommodating the eye its refractive power is increased enough to focus these rays on the retina and the near object becomes distinct The normal eye must accommodate +3 D to read at 33 cm Accommodation involves muscular work and nervous strain

Accommodation is measured by bringing fine print up to the eye of a person who wears his required distance correction The distance from the eye at which the object becomes *blurred is noted in centimeters One hundred is divided by this and the quotient is the accommodative power* in diopters

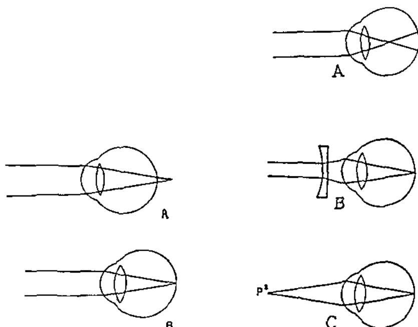


Fig 288—Hyperopia *A* With accommodation relaxed *B* Effect of accommodation on parallel rays*

Fig 289—Myopia *A* Parallel rays cross in front of retina *B* Effect of concave lens on parallel rays *C* Divergent rays from the punctum P² focus on retina without the use of accommodation*

ABNORMALITIES OF REFRACTION AND ACCOMMODATION

Errors in refraction or accommodation constitute the commonest causes for *asthenopia (eyestrain)* Either of these difficulties results in failure of the visual image to focus on the retina producing both visual and distant symptomatology which may be incapacitating to minor or major degree

ERRORS OF REFRACTION (AMETROPIA)

With normal refraction (emmetropia) the image is focused on the retina Abnormal refraction is productive of ametropia which may be of several varieties In far sightedness (*hyperopia*) the image is focused behind the retina whereas in near sightedness (*myopia*) the image is focused

* Gifford Textbook of Ophthalmology

vision of each eye Similarly the left optic tract consists of fibers from the left half of each retina or the right half of the field of vision of each eye

The optic tracts run laterally and backwards sweeping around the cerebral peduncles (crura cerebri) to end in two roots The lateral and larger root passes to the lateral geniculate body the pulvinar of the thalamus and the superior colliculus The smaller medial root goes to the medial geniculate body All the visual fibers are contained in the lateral root

The Lateral Geniculate Body—The lateral geniculate body receives the great majority of the visual fibers of the optic nerve which are relayed from it to the occipital cortex as the geniculo-calcarine pathway

The Superior Colliculus—The fibers that terminate in the superior colliculus are relayed to the nuclei of nerves III IV and VI They probably include pupillary fibers to the Edinger Westphal nucleus There are no efferent cortical fibers

The Pulvinar of the Thalamus—The pulvinar has apparently no part in the visual process in man but is concerned with ocular movements and stereognosis

The Visual Cortex—The geniculo-calcarine pathway makes its way through the temporal lobe and through the posterior portion of the internal capsule to form the optic radiations which end in the *area striata* of the occipital lobe surrounding the *calcarine fissure* The

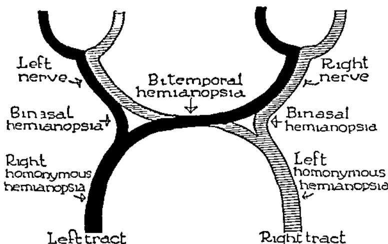


Fig 290—Hemianopsia

right visual cortex is concerned with visual impulses from the right half of each retina or the left field of vision

The Blood Supply to the Visual Pathways—The *chiasm* is supplied by branches from internal carotid anterior cerebral and anterior and posterior communicating arteries The *tract* is supplied by vessels derived from the posterior communicating and internal carotid arteries The *external geniculate body* is supplied by the posterior cerebral artery as is the main portion of the optic radiations The *visual cortex* receives its nourishment from the calcarine and other branches of the posterior cerebral artery

HISTORY

The patient with disturbances of the visual apparatus may complain of distant or local disturbances The more frequent remote complaints include headache (p 1512) and vertigo (p 2020) The more direct phenomena include the complaints of failing vision (p 1638) eyestrain (p 1537) conjunctival discharge (p 1619) excessive tearing (p 1525) photophobia (p 1574) and pain in the eye (p 1582)

A family history of similar difficulties suggests the presence of cataracts glaucoma myopia or the allergies A past or present history of

formed of two prisms whose apices are together in the center concave lenses diverge parallel rays of light

Cylindrical lenses or *cylinders* are segments of a cylinder parallel to its axis Light passing in the meridian or plane of the axis is not refracted and passes through unchanged as through a pane of glass Light passing through in a plane opposite or *perpendicular meets the curved surface* of the lens and is refracted The rays are converged or diverged depending on whether the cylinder is convex or concave It is necessary to indicate the axis or zero direction of a cylinder in an optical prescription

The Numeration of Lenses—The distance between the lens and the point at which it brings parallel rays to a focus is the *principal focal distance* The stronger the lens the shorter is this distance the strength of a lens is thus reciprocal to its focal distance The metric or dioptric system is used to classify lenses A lens whose principal focus is one meter distant is a 1 00 diopter (D) lens If it is stronger and has a focal distance of 0 5 or 0 2 meters it is a 2D or 5D lens respectively If it has a focal distance of 4 meters it is a 0 25D lens The spherical or cylindrical lens is a simple lens one that is both spherical and cylindrical is a *compound lens*

Contact Lenses—Contact lenses are thin shells of transparent glass or plastic material designed to be worn over the anterior portion of the eye ball They consist of a central portion which overlies but does not touch the cornea and a peripheral part which rests upon the sclera The space between the cornea and the contact lens is filtered with saline or isotonic buffer solution By its very nature corneal astigmatism is eliminated and *corrections for ametropia are ground into the glass*

The fitting of contact lenses is difficult The best and most improved method is to make a mold of the cornea and adjacent sclera under local anesthesia and to use this mold for casting the contact lenses

Contact lenses are of greatest value in keratoconus irregular astigmatism high myopias and for actors actresses and athletes The major objections to their use are the expense and the fact that the patient can tolerate them for only a number of hours

Protective Lenses—Tinted lenses are used for patients who complain of glare for workers exposed to bright reflected or fluorescent light and during exposure to strong sun or ice glare Sunglasses should be made of carefully polished lenses as inferior products contain irregular amounts of refractive strength and cause asthenopia

THE VISUAL PATHWAYS

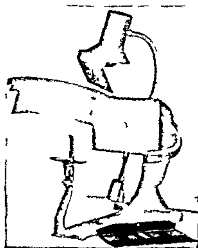
The visual process begins in retinal rods and cones The light impulse is transmitted to *ganglion cells of the retina* whose axones continue in the nerve fiber layer converging to the *optic disk* Here they form the *optic nerve* which terminates intracranially at the *optic chiasm*

The Optic Chiasm—The optic chiasm is formed by the junction and partial decussation of the two optic nerves It is a transversely oval body 30 x 8 x 4 mm and usually lies behind the tuberculum sellae although its position may vary to some extent *It may be* above in front of or behind the *pituitary body* (p 115?) The inner fibers of the optic nerves decussate in the chiasm

The Optic Tracts—*Axones* emerge from each posterolateral aspect of the chiasm to form *the optic tracts* Each optic tract contains fibers from each eye The right is made up of non-decussating fibers from the right or temporal half of the retina of the right eye and decussating fibers from the right or nasal half of the retina of the left eye Thus the right optic tract contains fibers from the right half of each retina or the left half of the field of

Examination for Visual Acuity —Visual acuity is tested by the *chart* previously described (p 1535) If there is any abnormality the ophthalmologist performs *refraction* (p 1535) In a patient below the age of forty five a cycloplegic is employed if there is any question of the visual acuity The patient is tested with and without correction

Retinoscopy permits the ophthalmologist to estimate the refractive error by noting through the retinoscope the movement of a shadow like reflex in the eye This procedure which is particularly accurate when the eye is dilated with a cycloplegic and mydriatic is the best method of estimating the refraction in children or in those adults who are mentally deficient When the shadow like reflex has been observed the ophthalmologist tries lenses before the eyes and notes the reversal of direction when the proper lens is inserted Unlike refraction retinoscopy does not require the cooperation of the patient and is an objective procedure



Fi 931 —Simplified Ferree-Rand perimeter *

Examination for Color Sense —The ophthalmologist uses the pseudoisochromatic chart of Stilling or Ishihara to provide a rapid and accurate differentiation of the color blind These charts are invaluable in excluding malingerers

Examination for Light Sense —The adaptometer used for determining light sense is an expensive instrument that is not very accurate Few ophthalmologists use the machine since the light sense may be evaluated by questioning the patient concerning the length of time that it takes to find a seat in the movies when he passes from a light lobby into a darkened theater and the effect in night-driving of the headlights of oncoming automobiles

Examination for Visual Fields —In estimating and plotting the visual fields the ophthalmologist performs a preliminary confrontation test (p 3627) in the same manner as the practitioner For more detailed work a *perimeter* is employed This instrument is a metallic semicircle repre

* Clark in Pullen, Medical Diagnosis.

hypertension 'kidney trouble' diabetes tuberculosis or syphilis give intimation that the ocular manifestation may be a local consequence of a systemic disturbance The previous use of quinine salicylate arsenicals reducing drugs (dinitrophenols) worm medicines' and laxatives containing phenolphthalein directs attention to possible drug idiosyncrasy or poisoning Ocular lesions which date to birth are most likely congenital anomalies or infections resulting from gonorrhea or neonatal syphilis At the extremes of life disturbances of the visual tract are most commonly due to presbyopia cataract or glaucoma

METHODS OF EXAMINATION

The practitioner is required to make a careful survey of the visual apparatus He inspects the eye by *oblique illumination* (p 3622), he tests visual acuity roughly with *reading charts* the color sense is approximated by *descriptions of objects in the room* dark adaptation becomes apparent from the *history* the visual fields are grossly marked out by the *confrontation test* (p 1541) In our opinion *ophthalmoscopy* (p 3628) is as much a part of the routine physical examination as auscultation and percussion

THE OPHTHALMOLOGIST

The positive indications for reference to the specialist ophthalmologist are

1 *Abnormalities* of the uveal tract (p 1632), the retina, media optic nerve or any of the visual functions (p 1638)
2 Investigation of *headache* (p 1512)
3 Investigation of *increased intracranial pressure* (p 1468)
4 Suspicion of the presence of a *brain tumor brain abscess* or trauma to the cranium such as a *fracture concussion laceration* of the brain or *intracranial bleeding*
5 *Meningeal irritation* (p 1462)
6 In *cardiovascular-renal disorders* (p 2379)
7 In *chronic disease* such as *tuberculosis* and *syphilis*
8 Exact determination of *visual acuity* (p 1541)
   (*a*) Charts
   (*b*) Refraction
   (*c*) Retinoscopy
9 Examination for *color sense* (p 1535)
10 Examination for *light adaptation* (adaptometer) (p 1535)
11 Examination of *visual fields* (p 1541)
   Perimetry stereocampimetry and angioscotometry
12 Examination for *ocular dominance* (p 1527)
13 Irrigation of *lacrimal passages* (p 1557)
14 Transillumination *of the orbit* (p 3632)
15 The use of the *slit lamp* (p 1544)
16 *Gonioscopy* (p 1545)
17 *Tonometry* (p 1545)
18 *Ophthalmoscopy* (p 3628)
19 *Exophthalmometry* (p 1546)
20 *Tests for squint* (p 1529)

screen is a large black cloth marked off with circles indicating the number of degrees of deflection from the central line of fixation of the eye The patient sits at a distance of 1 to 2 meters from the screen and focusses on its center A small dense object no more than 1 to 2 mm in size is employed and any defect (scotoma) in the central field is noted and measured A more elaborate device is the *campimeter* which is smaller and used at a lesser distance from the eye The *stereocampimeter* permits the patient to be examined with both eyes open at the same time The meas

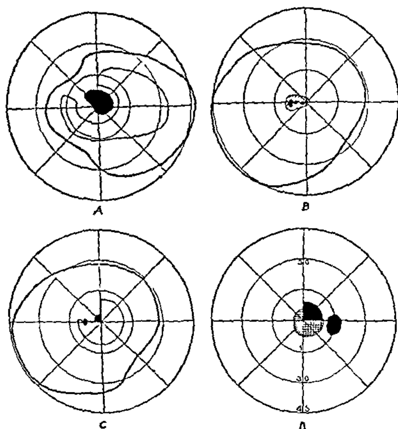


Fig 293—*A* Central scotoma retrobulbar neuritis *B* Paracentral scotoma with nuclei tobacco amblyopia *C* Uniocular quadrantic central scotoma junction scotoma *D* Chiasmal scotoma with quadrantic differentiation

urement of the size of the scotoma produced by the retinal vessels is termed *angioscotometry*

Tests for Ocular Dominance—In children who complain of reading difficulties the determination of the dominant eye is sometimes of value The ophthalmologist tests this by having the patient look into the wider end of a cardboard cone shaped like a megaphone The patient fixes upon an object in the distance Although apparently looking with both eyes due to the narrowness of the small end of the cone the object is fixed

Traquair in Berens The Eye and Its Diseases

senting 1 meridian of the hemisphere The apparatus can be revolved so that each meridian may be successfully studied The patient places the chin in a rest and *focusses one eye upon a central focal point* The other eye is closed The examiner then uses white and colored objects of known size to chart the exact limits of the visual field

The normal visual field is expressed in relative terms since it varies *within limits depending upon the intensity and size of the stimulus* employed Using a 3 mm white object on a perimeter of a 330 mm radius the peripheral limits of the field approximate 95 degrees outward 75 degrees downward 60 degrees inward and 60 degrees upward It is not uncommon to encounter a smaller field with a restriction of 10 degrees in

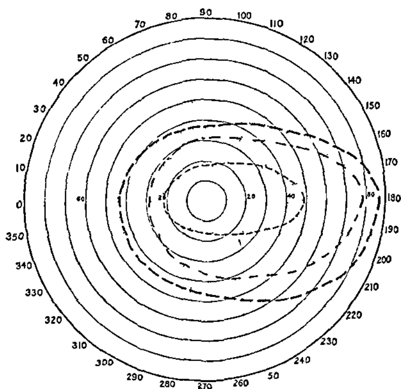


Fig 902—Fields for 10/330 blue red and green Note the horizontal elongation of the color fields Average of six subjects *

all meridians The field for color is smaller than that for white but has the same general contour Red is 10 degrees less than blue and green 10 degrees less than red

Normally there is present *a physiological blind spot which corresponds* to the penetration of the retina by the optic nerve The center of the blind spot is about 12 to 15 degrees outside of the fixation point and 1½ degrees below the horizontal meridian It measures approximately 7½ degrees in height and *5½ degrees in width*

The central 30 degrees of the visual field are studied against a black background by means of a tangent screen or a campimeter The tangent

* Traquair in Berens The Eye and Its Diseases

(floaters) an aqueous ray or translucent beam is seen Early and subtle changes in the iris fine particles in the retro lental space and the localization of lenticular opacities in early *cataract* (p 1592) can be observed accurately with the slit lamp

Slit lamp study of the *vitreous* (p 1544) is sometimes unreliable but definite changes are noted in *hemorrhage* and long standing *inflammation*

Gonioscopy —The details of the angle of the interior chamber are studied by using a special contact glass placed over the eye strong illumination and a magnifying device such as the slit lamp corneal microscope or an instrument known as the gonioscope These methods of ex

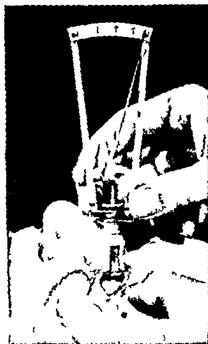


Fig 295—Tonometer of Schiotz being placed on the cornea

amination are useful in glaucoma tumors of the iris and ciliary bodies and in localizing foreign bodies

Tonometry.—For a more accurate measurement of the intra ocular tension (p 1526) the ophthalmologist uses a tonometer which records the resistance offered by the anesthetized cornea to a definite weight It does not measure directly the intra ocular tension but is calibrated against an experimental setup in which actual intra ocular readings are recorded The limits of normal with the Schiotz instrument are considered to be between 15 and 29 mm of mercury In some people 29 is higher than the eyes can tolerate while others may tolerate far more without any ocular deterioration See *Glaucoma* (p 1578)

Ophthalmoscopy —The ophthalmologist uses the direct ophthalmoscope as does the practitioner While atlases are available with plates of the

G fford Textbook of Ophthalmology

with only one eye The eye that does the looking is the dominant eye Most right handed individuals have a dominant right eye Many children *with reading difficulties have dominance of the left eye* usually associated with left handedness Retraining treatments consist of compelling the patient *to use the right eye* if necessary occluding or covering the left

Irrigation of the Lacrimal Passages—In order to determine patency of the lacrimal passages the ophthalmologist irrigates fluid by means of a blunt lacrimal needle into the canaliculus If the fluid flows unimpededly into the nose or throat there is no significant obstruction This procedure is painless in contradistinction to probing of the lacrimal passages

Transillumination—The ophthalmologist uses the ophthalmoscope light as a transilluminator The head of the apparatus is detached and the bulb is placed deep in the orbit through the lid or anesthetized conjunctiva The *pupil is observed for obstruction of light reflection by a tumor vitreous* hemorrhage or neoplasm Transillumination is also used to demonstrate *atrophy* of the *iris* In this condition fine points of light become evident in the iris during the procedure

The Slit Lamp—By means of the slit lamp the ophthalmologist obtains a magnified binocular view of the front half of the eye The slit lamp



Fig 931—Optic section through the eye produced by the narrow beam of light of the slit lamp *H* cornea *I* anterior chamber *L* lens *G* vitreous

consists of a binocular microscope magnifying objects twenty or forty times A special light projects a sharply defined focussed beam into the eye The beam traverses the part to be examined showing it in optical section while the remainder of the eye remains in darkness The lighting system is very flexible and the light can be so directed as to give direct focal illumination retroillumination or specular reflection The width of the beam may be widened or narrowed as desired The method permits the magnification of the area or tissue and localization of lesions according to depth

The slit lamp shows all types of *inflammation of the cornea* (p 1626) involving the epithelial layers and the deeper structures Vascularization an important indication of certain types of inflammation is apparent by retro illumination The appearance and size of the corneal nerves and the character of the endothelium of the cornea can be noted Finely pigmented or white precipitates may be seen on the posterior surface

In *iritis* (p 1633) when the aqueous contains fine floating particles

Alfred Vogt

A *mononuclear response* in the epithelial scrapings suggests epidemic virus keratoconjunctivitis or the milder Beal type of conjunctivitis in vernal conjunctivitis *eosinophilia* is demonstrable inclusion bodies are found in *trachoma* and *inclusion conjunctivitis* Giemsa stains are particularly valuable in the demonstration of pathogenic cocci

BIOPSY

Biopsy of the conjunctiva may reveal the presence of a *leptothrix infection* as in *Parinaud's conjunctivitis* It is also of value in the recognition of *tumors*

TREATMENT OF OCULAR DISORDERS

The practitioner can make considerable progress in the treatment of most ocular disorders by external applications methods of physical therapy and minor surgical procedures More serious ophthalmologic disorders are best referred to the specialist

LOCAL APPLICATIONS

Local applications are applied to the lids and eyes for purposes of cleansing (collyria) protection pupilloconstriction pupillodilatation local anesthesia bacteriostasis and bactericidal effects

DIFFERENTIAL DIAGNOSIS OF OCULAR DISORDERS

The differential diagnosis of ocular disorders has been tabulated in several places with reference to the presenting symptoms or signs The most important of these are *Epiphora (Tearing)* (p 1525) *Diplopia (Double Vision)* (p 1528), *Mydriasis and Miosis* (p 1533) *Abnormalities of the Pupils other than Mydriasis and Miosis* (p 1534) *Disturbances of Color Light and Form Sense* (p 1535) *Photophobia* (p 1574) *Exophthalmos (Proptosis)* (p 1575) *Papilledema (Choked Disks)* (p 1579) *Pain in the Eye* (p 1592) *Disturbances of the Eyelashes and Eyelids* (p 1612) *Disturbances of the Orbit* (p 1615) *Disturbances of the Conjunctiva* (p 1627) *Alterations in the Acuity of Vision (Amblyopia Amaurosis Dimness of Vision and Blindness)* (p 1638), *Ptosis (Lid Lag)* (p 1649) *Disturbances of the Fields of Vision (Hemianopia and Hemianopsia)* (p 1645) *Nystagmus* (p 1534)

PHYSICAL THERAPY FOR THE EYE

Physical therapy for the eye is accomplished by heat and cold infra red ray diathermy roentgen ray hyperthermia grenz rays phototherapy massage and iontophoresis

Heat and Cold —Heat causes active hyperemia absorption of products of inflammation and increase in local antibodies *Hot moist compresses* are best applied with pieces of cotton or cloth wrung out in water as hot as can be tolerated They are placed upon the closed lids and renewed every minute Running hot water from a washstand is as serviceable as boric acid Compresses are useful in inflammations of cornea ciliary body iris sclera and orbit They hasten the formation of pus and relieve pain in hordeola incipient chalazia lacrimal abscess and panophthalmitis Heat is contraindicated in conditions accompanied by intra ocular hemorrhage

Cold produces constriction of small vessels and lessens swelling which

commoner abnormalities the importance of positive findings is such that the specialist should be consulted for confirmation of the ophthalmoscopic picture and its interpretation A binocular view of the fundus is obtained by the *Gullstrand ophthalmoscope* a large expensive instrument not in general use

Exophthalmometry —*Several methods are available to the specialist for* the measurement of *proptosis* or *exophathalmos* (p 1575) The most common procedure is the use of the Hertel exophthalmometer This instrument is placed against the sides of the orbit and the protrusion of the eyeball is measured on a millimeter rule by a system of mirrors The intra orbital distance is noted for subsequent comparative examinations

The normal readings are between 16 and 18 mm at 96 to 100 intra orbital distance A proptosis of 20 is definitely pathological Increasing readings are of more value than a single observation See *Hyperthyroidism* (p 1197)

Recording Eye Movements —Using a special machine eye movements may be recorded This is of value in those who have reading difficulties and is often an initial step in the reeducation of patients with reading problems

LABORATORY EXAMINATIONS IN DISEASES OF THE EYE

Because the eye frequently reflects local manifestations of systemic disturbances (p 1598), it is essential to perform complete laboratory examinations in the presence of any ophthalmologic disorder These must include *serologic tests for syphilis hemogram urine analysis* and if indicated a *tuberculin test* and a *chest radiograph* With exophthalmos a *basal metabolic determination* is required With *papilledema roentgenologic examination of the skull lumbar puncture* and a review of the complete *neurologic status* become mandatory

BACTERIOLOGY OF THE EYE

Bacteriologic investigations of the eye often yield important information *Cultures* are made by briskly rubbing the conjunctiva of the lower lid with a sterile cotton applicator which has been moistened with glucose broth The material is immediately inoculated on a *blood agar plate* Using separate swabs the lid margin is treated similarly to culture hair follicles and glands Culture of a corneal ulcer is accomplished with a platinum loop After the agar plate has been inoculated secretion is smeared on a glass slide and stained by the Gram method for immediate examination Recognizable pathogens include staphylococci streptococci pneumococci meningococci gonococci H influenzae H conjunctivitidis (Koch Weeks) H duplex (Morax Axenfeld) B diphtheriae P tularensis and M mallei (Glanders)

EPITHELIAL SCRAPINGS

Information of value is obtained from epithelial scrapings of the conjunctiva A platinum spatula is employed after local anesthetization The material is placed on a slide fixed with methyl alcohol for five minutes and stained with dilute Giemsa (1 drop to 2 cc of distilled water) The stain is kept on overnight at room temperature or for one hour in the incubator To bring out inclusion bodies the slides are passed momentarily through 95 per cent ethyl alcohol

a layer of gauze Cold compresses are especially useful in conjunctiviti (p 1616) Cold may be used in preference to heat in acute congestive glaucoma In iritis heat may aggravate pain and the use of cold for short periods is comforting

Infra red Rays —The infra red lamp is a simple and clean means of providing heat to the eye Applied to the closed lids no damage is done by infra red therapy Special lamps have been designed for eye work

Infra-red light from 13 000 to 16 000 Angstrom units is absorbed almost entirely by the cornea Large doses of infra red may produce cataract a possible explanation of the high incidence of this condition in India Large doses may cause permanent injury of the retina as illustrated by the macular lesion in solar eclipse blindness

Medical Diathermy —Short wave and electromagnetic diathermy have been tried on the eye Short wave diathermy may raise the temperature in the vitreous as high as 109° F The effect seems useful in *orbital cellulitis* and *chronic uveitis* Elevation of intra ocular temperature above 106° F has been reported to cause *thrombosis of the central retinal vein*

Surgical Diathermy —Surgical diathermy is most widely used in the operation for *retinal detachment* for permanent removal of cilia (often done by electrolysis) for correction of *entropion* and *ectropion* (Ziegler cautery operation) and for removal of small *chalazia* or *papillomas* of the lids In large *orbital tumors* electrocoagulation blocks avenues of extension and reduces bleeding

The Thermophore —The thermophore is a device designed to apply a measured amount of heat (145° F) directly to an ulcer of the cornea for a definite period of time usually one minute

Roentgen Ray and Radium Treatment —Roentgen therapy lies entirely in the province of the roentgenologist as cataracts damage to the lids and cornea and glaucoma may follow improper use The use of x rays or radium is indicated in the more diffuse types of *skin neoplasm* especially basal cell epitheliomas and those adherent to bone *Vascular nevi* about the lids are especially suited to radium treatment *Intra ocular tumors* do not respond very well to irradiation therapy but in conditions such as *bilateral gliomas* (p 1419) it is the only means available other than removing both eyes

Early deep hordeolum acute dacryocystitis early post traumatic or postoperative intra-ocular infection chronic blepharitis tuberculous iridocyclitis and choroiditis and thrombosis of the central vein of the retina may respond favorably to roentgen therapy

Grenz Rays —The border rays described by Bucky are between the ultraviolet and the longer roentgen rays in the spectrum measuring about 2 Angstrom units These rays have slight penetrating power and are believed incapable of producing the deleterious effects of roentgen rays Their efficacy is under investigation for the treatment of superficial corneal lesions and inflammations of the lids

Phototherapy —Local phototherapy with ultraviolet light is delivered by the Birch Hirschfeld lamp Its popularity has waned with the advent of other means of therapy and its main usefulness if any seems to be in *dendritic keratitis* and *superficial catarrhal ulcers*

The cornea absorbs almost all the ultraviolet rays below 2950 Angstrom units and the lens absorbs all the higher ultraviolet rays Corneal absorp-

accompanies early stages of infection or reactions to trauma Cold is best applied to the closed lids with pieces of cotton frequently changed from

TABLE 105—LOCAL APPLICATIONS OF LIDS AND EYES

Collyria	Physiological Saline 4 per cent Boric Acid
Astringent	0.2 to 0.3 per cent Zinc Sulfate or Copper Sulfate added to collyrium
Vasoconstrictor	Epinephrine 1 1000 added to collyrium in ratio of 1 10
Rubefacient	Ethylmorphine Hydrochloride (dionin) 5 to 20 per cent solution
Local anesthetic	Pontocaine ½ to 1 per cent Butyn 1 to 4 per cent Metycaine 2 to 4 per cent Holocaine (Phenacaine) 1 per cent Cocaine Hydrochloride 1 to 5 per cent Procaine 2 per cent
Protective	Petroleum Jelly Castor Oil
Bactericide	1 3000 to 1 10 000 Bichloride of Mercury Ointment for lids 2 to 3 per cent Ammoniated Mercury Ointment for lids 1 per cent Yellow Oxide of Mercury for lids 1 to 2 per cent Zinc Oxide Ointment for lids 5 per cent Sulfathiazole Ointment for lids Sulfanilamide Powder 30 per cent Sodium Sulfacetimide 2 per cent Tincture of Brilliant Green in 70 per cent alcohol for lids 10 per cent strong Silver Proteinate 25 per cent weak Silver Proteinate ¼ to 1 per cent Silver Nitrate 2 per cent Mercurochrome 1 or 2 per cent Ethylhydrocupreine Hydrochloride (optochin) Tyrothricin (0.3 per cent) Penicillin (1 cc equals 250 to 2000 units)
Miotics	1 to 2 per cent Pilocarpine Hydrochloride 0.5 to 1 per cent Physostigmine Salicylate (eserine) 2 to 5 per cent Neostigmine 5 to 20 per cent Acetylcholine 1 to 20 per cent Mecholyl 0.5 to 2 per cent Doryl 0 1 per cent Diisopropyl Fluorophosphate (DFP)
Mydriatics	5 to 10 per cent Eucatropine (Euphthalmine) ¼ to 3 per cent Atropine Sulfate 1 to 2 per cent Homatropine Hydrobromide 0 1 to 0.5 per cent Scopolamine Hydrobromide 1 per cent Amphetamine Sulfate ¼ to 2 per cent Neosynephrine 1 per cent Paredrine 3 per cent Ephedrine Sulfate 2 to 5 per cent Cocaine Hydrochloride
For staining cornea	2 per cent Fluorescein in 3 per cent Sodium Bicarbonate

a bowl of cracked ice Ice should never be applied directly to the lids If sterility must be observed cracked ice in a rubber glove is pl[illegible] over

orbit or globe *Diphtheria antitoxin* (p 310) is used in the rare instances of conjunctivitis due to *B diphtheriae* and has also been recommended in large doses (10 000 to 16 000 units) for *sympathetic ophthalmia* and in smaller doses (5 000 units) for *herpes zoster ophthalmicus* *Antimeningococcic serum* was used extensively in the ocular complications of epidemic meningitis especially endophthalmitis Since the advent of antibiotics its value has declined although it may be wise to combine the methods *Antipneumococcic serum* has been tried in ulcers of the cornea but is being superseded by sulfonamides *Transfusions of whole blood* from persons who have recovered from herpes zoster seem to prevent ocular complications and mitigate serious consequences after the disease is established

Vaccines—In ophthalmology the chief use of vaccines has been in the treatment of recurrent hordeola and severe blepharitis due to *staphylococci* *Autogenous vaccines* prepared so as to leave the bacterial proteins practically unchanged seem best It is advisable to start treatment with a minute intradermal injection (0 02 cc) to judge the patient s sensitivity to the toxins or bacterial proteins Later subcutaneous injections are given in amounts causing no marked local and certainly no focal or ocular reaction (p 80)

Gonococcal vaccine seems useful in gonorrheal iritis (p 1633) Autogenous vaccines prepared from foci of infection that cannot be entirely eradicated sometimes are of great benefit in corneal ulcers and uveitis

TOXOID—The importance of the exotoxins of staphylococci in the production of chronic blepharitis conjunctivitis and keratitis has stimulated the use of *staphylococcus toxoid* or weakened toxin in the digest modified form (Lederle) Dosage is based on the reaction to small intradermal injections (0 001 to 0 005 cc) Semi weekly injections are given with increasing doses (0 1 cc) depending upon the responses The maximal amount is 1 0 cc

Tuberculin—Tuberculin therapy is extensively used in ophthalmology especially by those who are unimpressed by the importance of focal infection In contradistinction to its uselessness in pulmonary tuberculosis it is considered an important factor in the treatment of *ocular tuberculosis* especially *sclerosing keratitis iritis iridocyclitis* and *chorioretinitis* In these special manifestations of the disease frank pulmonary tuberculosis is seldom present If active tuberculosis is present elsewhere in the body tuberculin therapy is contraindicated

TECHNIC—A *diagnostic series* of intradermal injections is carried out with very small doses of old tuberculin In a severe case the initial injection is a control of saline followed by 0 000001 mg of O T The reaction is read at twenty four and seventy two hours If negative stronger doses of 0 00001 mg and 0 0001 mg are tried Positive tests are of little diagnostic value but indicate the proper dosage for the initiation of therapy

Treatment with tuberculin is always of long duration particularly if an initially high sensitivity precludes all but minute doses at the onset Injections are usually given twice a week being gradually increased until 1 to 5 mg are tolerated without a reaction When a local reaction is encountered the next injection is the same as the previous dose which caused no reaction When a dose of 0 1 mg is reached the intervals are lengthened to one week

tion after exposure to ultraviolet light results in an *abiotic reaction* and an extremely painful *keratitis* Similar mechanisms are involved in *photophthalmia* or *snow blindness* and after accidental exposure to the flash of an *electric arc* *Treatment* consists of the instillation of epinephrine and a local anesthetic after which dark glasses are worn

Massage —Massage is useful in clearing the opacity that follows corneal ulceration in the maintenance of normal intra ocular tension in glaucoma especially after a filtering operation and in the treatment of *meibomitis* and *chalazia* (p 1611) In corneal ulceration, 2 per cent boric ointment is anointed and the eye is gently massaged by rubbing the closed lids with the forefinger In *glaucoma* the two index fingers are placed on the upper lid of the depressed eyeball each finger alternately is pressed in and released so as to indent the globe The massage is done thirty times and repeated two or three times a day

Iontophoresis or Galvanic Therapy —Using a dry cell battery (galvanic electricity) in a special apparatus medication can be administered to the eye in an ionized form increasing the concentration of the drug in the aqueous Iontophoresis with sulfanilamide and penicillin is of great help in corneal ulcers due to *B pyocyaneous* a condition in which treatment hitherto has been unsatisfactory as well as in conjunctival and other superficial infections

SYSTEMIC METHODS OF THERAPY

The useful methods of systemic therapy in ophthalmologic disorders include ultraviolet exposure elimination of foci of infection the administration of immune serums toxoids vaccines and tuberculin non specific foreign protein therapy hyperthermia specific chemotherapy with anti luetic drugs sulfonamides and the antibiotics such as penicillin and the use of vitamins and hormones

Ultraviolet Therapy —General ultraviolet irradiation produces a mild skin reaction and changes in the blood and tissues that resemble those of foreign protein shock *Tuberculous uveitis sclerokeratitis* and *phlyctenulosis* seem to be influenced favorably by irradiating one third of the body surface at one sitting and repeating three times a week for fifteen treatments General ultraviolet therapy has also been suggested for the treatment of *sympathetic ophthalmia*

Elimination of Foci of Infection —In ocular inflammations particularly those involving fibrous tunic and uveal tract (iritis iridocyclitis and choroiditis) a careful search is made for foci of infection *Tonsillar* and *nasal infections* play an important role in optic neuritis and in uveitis of younger people In older people *dental sepsis* is particularly important as a cause for uveitis and keratitis Dead teeth and retained roots are suspect and devitalized teeth require peri apical exploration even if roentgenograms are normal The *genito urinary tract* deserves investigation The possibility of an *intestinal focus* of infection must be considered

Biological Therapy —Biological therapy employs serums vaccines toxoid tuberculin and foreign protein

Serums—Immune serums are used only occasionally in ophthalmic treatment *Tetanus antitoxin* (p 297) is indicated after an injury in which material likely to contain tetanus spores is carried into the lids

phopathia venereum It also merits trial in Welch bacillus infection in uveitis of unknown origin and in Koch Week s conjunctivitis Little success has been experienced however in Morax Axenfeld infection or epidemic virus conjunctivitis

Untoward ocular reactions that follow systemic sulfonamide therapy include transient myopia edema of the lids chemosis and allergic conjunctivitis retinal hemorrhages optic neuritis and mydriasis

Penicillin—Penicillin like sulfonamide may be used locally or systemically in ophthalmic infections For topical application it is employed in a strength of 250 to 10 000 units per cc with concentrations augmented by iontophoresis (p 3792) Penicillin may be injected directly into the aqueous by the ophthalmologist The systemic introduction is accomplished by intramuscular or intravenous injection (p 106)

Penicillin has certain advantages over the sulfonamide drugs despite the fact that local sensitivity to the ointment may occur infrequently Direct injections of penicillin into aqueous are highly recommended in penetrating wounds and metastatic abscess It is also worthy of trial in anaerobic diphtheritic and spirochetal invasions The effects of systemic administration in gonorrheal ophthalmia are truly miraculous

In the treatment of coccal infection there seems no question as to the lesser irritation and better results of penicillin as compared to sulfonamide Ambulatory patients must be treated by topical instillation but those who are hospitalized or accessible to nursing service may be given intramuscular injections In the virus infections penicillin is inferior to the sulfonamides particularly sulfanilamide

Penicillin has prophylactic value in the management of ocular injuries and in intra ocular surgery It is given in large doses in orbital cellulitis where parenteral administration is supplemented by topical application of a solution containing as high as 10 000 units to the cc A suitable ointment for the treatment of blepharitis styes and furuncles is improvised by the addition of 250 to 500 units to each gram of 25 per cent petroleum jelly with 75 per cent cold cream

Tyrothricin—Concentrations of 5 to 33 mg per 100 cc of tyrothricin appear to be effective against gram positive organisms particularly pneumococci staphylococci and streptococci The suspension can only be employed locally since systemic administration is fraught with danger

Streptomycin—Streptomycin locally or systemically may prove of great value in the treatment of conjunctivitis due to *H influenzae* Koch Weeks and Axenfeld bacilli *P tularensis M tuberculosis* and *M leprae*

Hormone Therapy—Thyroid extract (p 1189) has been employed in myopia keratoconus and chronic uveitis without any definite evidence of success In parathyroid tetany which is often accompanied by cataracts the early administration of parathyroid hormone (p 1223) may prevent the development of cataracts or stay their progress if lens opacities have already developed Insulin (p 1237) is of value in the treatment of ocular complications of diabetes Despite enthusiastic claims of its value in glaucoma and myopia cortin injections (p 1267) have proved most disappointing

Vitamin Therapy—Vitamin A is definitely indicated in keratomalacia a true nutritional necrosis of the cornea It also is recommended for kerato

Foreign Protein—Foreign protein therapy has been successfully employed in the treatment of a variety of ophthalmic conditions including acute conjunctivitis gonorrheal ophthalmia, phlyctenular keratitis interstitial keratitis corneal ulcer acute and chronic iritis uveitis postoperative iridocyclitis scleritis sympathetic ophthalmia retinitis choroiditis optic neuritis retinitis pigmentosa herpes zoster and acute trachoma

Reactions may be produced by injections of boiled milk typhoid vaccine or the malarial parasite Milk is prepared by boiling for four minutes and cooling For the adult intramuscular injections of 8 to 12 cc are employed while 1 to 2 cc suffice for the infant With typhoid vaccine the H antigen is given in an initial intravenous dose of 10 000 000 and the amount is increased until a febrile reaction approximating 103° to 104° F has been accomplished In malarial therapy 5 to 10 cc of blood are taken from an infected donor suffering from the benign tertian variety The malarial blood is injected into the vein of the recipient who is then permitted to have 8 to 12 chills after which the paroxysms are terminated by the administration of quinine sulfate 0 6 gm (10 grains) given three times daily for five to seven days (p 517)

Hyperthermia—In addition to fever therapy using foreign protein typhoid vaccine and malarial parasites hyperpyrexia may be effected by the inductotherm or the hypertherm (p 3789) These technics are specialist province and are not to be attempted except under institutional supervision Principal indications are interstitial keratitis and optic atrophy of syphilitic origin

Specific Anti infective Therapy—Tremendous strides have been made in the anti infective therapy of ophthalmic infection The practitioner has access to sulfonamides penicillin streptomycin and tyrothricin The first three of these may be used topically or systemically tyrothricin however is only available for topical application

Sulfonamide—The available sulfonamide drugs for local application include 5 per cent sulfathiazole application of sulfanilamide powder and 30 per cent solutions of sodium sulfacetimide (p 88) The disadvantage of the local use of the sulfonamides is the frequent production of contact dermatitis of the lids allergic conjunctivitis increased tendency to opacification of the cornea delayed healing and increased scarring The advantages of local therapy are the lesser hazards of producing toxic systemic manifestations

The systemic use of the sulfonamides results in the appearance of the drug in the aqueous Sulfanilamide and sulfadiazine appear in the highest concentrations sulfapyridine is intermediate and sulfathiazole is least well excreted Those who favor the systemic method of therapy contend that benefits produced by topical methods are dependent upon absorption into the general circulation with re excretion into the aqueous They do not believe that bacterial antagonism is produced as the result of direct action of locally applied drug

Whether used locally or systemically however there is general agreement that the great value of the sulfonamides is in the treatment of gonococcal pneumococcal meningococcal staphylococcal and streptococcal infection Sulfanilamide appears to be of demonstrable if not specific value in the virus infections of trachoma inclusion conjunctivitis and lym

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conjunctivitis sicca epithelial dystrophy recurrent catarrhal ulcers and asthenopia associated with photophobia

Thiamine chloride appears to be of great value in toxic amblyopia due to alcohol and other forms of optic neuritis Deficiency of riboflavin is believed to cause a peculiar keratitis associated with vascularization of the cornea Rosacea keratitis is said to clear rapidly on adequate doses of riboflavin Its use is also suggested in attempts to influence the course of senile cataracts

Ascorbic acid is indicated in diabetic retinopathy intra ocular hemorrhages and the treatment of senile cataracts *Viosterol* is advised in phlyctenular keratitis but has no definite valuable effect in myopia

MINOR SURGERY

Much of the minor surgery of the eye must necessarily be done by the practitioner The more complicated procedures however merit reference to the specialist ophthalmologist

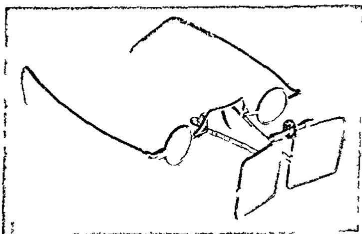


Fig 296 —Binocular magnifier *

Instruments —Procedures involving the eye require a *cilium forceps* and a *loupe magnifier to assist in the detection* and removal of foreign bodies (p 1572) in addition to the minor surgery kit

Application of Silver Nitrate to the Lids —After local anesthetization an applicator moistened with 1 per cent silver nitrate is rubbed briskly over the lid margin and cilia An attempt is made to remove scales and penetrate into the *underlying ulcerations To alleviate the sting* a drop of epinephrine is instilled into the conjunctival sac

Application of Silver Nitrate to the Conjunctiva —After anesthetization an applicator is moistened with 1 per cent silver nitrate and gently touched, but not rubbed on the conjunctiva of upper and lower lids After waiting several minutes the conjunctival sac is irrigated with salt solution If the eyes remain irritated the patient applies cold compresses for five minutes and a drop of epinephrine *is instilled to relieve residual* smarting

* Gifford Textbook of Ophthalmology

Application of Copper Sulfate in Chronic Conjunctivitis and Trachoma.—After anesthetization the copper sulfate stick is touched gently to two or three places on the lower lid. The eye is then treated as after the application of silver nitrate. Copper treatment is very irritating at first but the patient achieves a tolerance to repeated applications.

Manual Expression of the Lids.—Manual expression of the lids is of value in *blepharitis* (p. 1609), *meibomitis* (p. 1613) and *chalazia* (p. 1611) in their incipient stages. One thumb is placed on the lower lid, the other on the upper lid and the two lids are squeezed together to express secretion.

Removal of Cilia.—Removal of cilia is done for *trichiasis* (p. 1569), *distichiasis* (p. 1569) and the abortive treatment of *styes* (p. 1610). A special cilium forceps is required as lashes are so fine that they often slip through the best forceps. Breaking the lash in the middle may aggravate discomfort and make removal almost impossible.

Rupture of Cysts of the Lid Margin.—Cysts of the lid margin (p. 1566) may act as foreign bodies. They are easily ruptured with a wooden applicator or the tip of a sharp scalpel.

Removal of Foreign Bodies from the Upper Lid.—To evert the upper lid the patient is instructed to look down. The physician grasps the lashes with the left hand and pulls the lid up while exerting counterpressure with a cotton applicator on the upper lid just above the upper tarsal border (i.e., 7 to 8 mm. above the lid margin).

Once the lid is everted the foreign body (p. 1572) is usually apparent and is removed with a moistened cotton applicator. If none is seen, folds in the tarsus are explored with the applicator. Foreign bodies are not always black but may be any color so that any suspicious object, even if flesh-colored, is touched to see if it is removable. If *lithiasis* or *calcific yellowish deposits* (p. 1566) of the conjunctiva scratch the cornea, they must be removed in the manner of foreign bodies. After instillation of a few drops of anesthetic they are picked off with a sharp pointed knife or a hypodermic needle.

After removal of the foreign body *fluorescein* is instilled and washed out. Injured corneal epithelium stains green and this is sufficient reason to require the application of a dressing.

Application of the Eye Dressing.—When corneal epithelium is injured, a *patch* must be applied until the cells no longer stain with fluorescein. Sulfathiazole ointment (5 per cent) is instilled in the conjunctival sac and a piece of moistened gauze or woven cotton material is placed over the closed lids to prevent loose dry cotton, which is then placed on top of this, from scraping the cornea. The dressing is held in place by 1 inch adhesive strips or by Scotch (cellulose) tape. The adhesive is applied in a diagonal direction so that it ends on the malar bone and does not interfere with chewing. If a pressure dressing is desired, gauze is used instead of cotton and 1 inch wide strips of adhesive are applied.

If a *continuous wet dressing* is desirable, as in acute dacryocystitis or an abscess of the lid, the cotton is replaced by gauze moistened with penicillin, sulfanilamide, boric acid or saline. This may be covered with waterproof tissue and held in place by a 1½ inch bandage. The first few turns of the bandage are placed horizontally around the head under the occiput. They are then brought up under the ear to cover the eye, after which

they are wound around the head At the meeting of the diagonal and horizontal portions the bandage is tied with small vertical strips of gauze or adhesive, similar ties are made above and below the ear

Removal of Corneal Foreign Bodies—It is advisable to refer the patient with a corneal foreign body especially of the imbedded metallic type to the ophthalmologist If this is not feasible the patient is placed in recumbency and is ordered to fix both eyes on some landmark on the ceiling With adequate light best obtained by focal illumination and local anesthesia a black or white foreign body can usually be removed with a moist cotton applicator A brown appearance suggests that the imbedded metallic foreign body has oxidized or rusted and a fine *hypodermic needle* is required for removal Although it is necessary to remove the rust as completely as possible damage to the cornea may result in *destruction of Bowman's membrane* with resultant *permanent opacity* If all the rust is not removed liquefaction of the involved area may occur within a week or so the rusted portion then sloughs out and a serious ulcer is produced

After the foreign body has been removed penicillin solution is instilled sulfathiazole ointment is applied and the eye is covered with a patch In a rust ring foreign body there usually is considerable iritic irritation and mydriasis is accomplished with 2 per cent homatropine provided there is no evidence of increased intra ocular tension (p 1526) The dressing is changed daily until complete healing is evidenced by failure to obtain a staining reaction with fluorescein

Incision of a Hordeolum—Anesthesia is not necessary for external incision of a *hordeolum* (p 1610) Using a sharp pointed knife an incision is made horizontal or parallel to the lid margin where the lesion points Pus is evacuated by mild expression if necessary

Removal of a Chalazion—The area of the chalazion (p 1611) is blocked off proximally by infiltration through the skin with procaine The injection then is extended down to the lid margin The lid is everted a few drops of anesthetic are instilled into the conjunctival sac and additional procaine is injected subconjunctivally just proximally to the tarsus in the region of the chalazion A special chalazion clamp is applied to expose the lesion and render the area bloodless The chalazion is incised through the conjunctival surface of the lid by means by a vertical incision If it is firm the granulomatous tissue is dissected with a scissors otherwise it is scooped clean with a curette An attempt is made to save as much of the conjunctiva as possible The incision is best left unsutured If the chalazion points through the skin a horizontal incision is made through the skin which must be carefully sutured at the end of the operation A dressing is applied for one day unless the skin is sutured in which case it is maintained for three to four days Skin sutures are removed after four days

Repair of Lacerations of the Lid—Repairs of lacerations of the lids (p 1571) especially those involving the margins or associated with avulsion at the medial canthus are procedures for the specialist Suture of a laceration of the lid may be followed by subsequent deformity It is better to delay suture of a laceration than to do a hasty and improper approximation Careful apposition of fragmented pieces is required using numerous thin silk sutures Unless great pains are taken with a torn lid margin notching occurs Sutures tied on the conjunctival side of a through and

through lid laceration may cause damage and ulceration of the cornea The application of a firm pressure dressing is as important as the suture

Cauterization of Corneal Ulcers—After thorough anesthetization a fine applicator is moistened with a mixture of equal parts of *Tincture of Iodine* and *Glycerin* and applied to the ulcer until the area stains brown Applications of *carbolic* or *trichloracetic acid* are the province of the specialist Permanent opacification due to a chemical burn need not be feared from the iodine glycerin mixture which does not drip and run over Atropine drops (1 per cent) are applied an anesthetic ointment is spread on and a firm dressing is applied

OPHTHALMIC SURGERY

In this brief review of the scope of ophthalmic surgery only broadest principles are described for the guidance of the general practitioner

Probing of the Lacrimal Passages—Probing of the lacrimal passages is a specialist office procedure done for obstruction or stenosis of the lacrimal passages (p 156) Performed gently after preliminary instillation of 2 per cent procaine or 5 per cent cocaine in the lacrimal sac the procedure is not very painful One successful probing may relieve the condition If hemorrhage occurs when an adhesion is broken new adhesions may form and perpetuate the obstruction In infants with a congenital obstruction (p 1560) probing of the lacrimal passages under general anesthesia is generally very successful although the benefits may not be obvious for several weeks following the operation

Incision of the Lacrimal Sac—Incision of the lacrimal sac is done for *acute dacryocystitis* (p 1614) General anesthesia is required and after treatment is that of any other abscess

Dacryocystectomy—Extirpation of the lacrimal sac is performed under local anesthesia for *chronic purulent dacryocystitis* (p 1614) Hospitalization after the operation is brief The patient wears a dressing over the affected eye for one week This procedure removes a serious focus of infection and helps clear up tearing by the relief of coexisting conjunctivitis Persistent postoperative tearing is a serious objection to its performance Ophthalmologists are confining this procedure increasingly to older people

Dacryocystorhinostomy—In this operation instead of removal of the sac an incision is made in its medial wall which is then anastomosed to the nasal mucosa after a portion of the bony wall of the nose has been removed A permanent opening is established between tear sac and nasal cavity The procedure is done under local or general anesthesia and is rapidly becoming the method of choice except in people of advanced age It is a considerably more formidable procedure than dacryocystectomy and requires hospitalization for five to seven days Symptoms are alleviated and tearing stops in 80 per cent of cases

Extirpation of the Accessory Lacrimal Gland—If tearing continues after dacryocystectomy removal of the accessory lacrimal gland is advised The operation is done under local anesthesia

Plastic Operations on the Lids—Plastic operations on the lid include some of the simplest and most complicated procedures in ophthalmology The results are usually good except when extensive reconstructive surgery is required The latter are best performed by an ophthalmologist specializing in plastic surgery Even in the best hands however repeated failures occur time and again and the surgeon must have considerable patience to achieve a final good result

Ptosis Operations—Ptosis operations are usually performed on small children Proper procedures yield results that are usually eminently satisfactory

Keratoplasty—Transplantation of cornea have been successfully performed by specialists for restoration of vision in patients who e vision was lost due to extensive corneal opacities (p 1628) A graft of clear cornea is taken from an eye removed for another cause or from a cadaver specimen The graft usually takes and may remain clear enough to improve the patient vision considerably

Tattooing of the Cornea—A blind eye with an ugly obvious corneal opacity may be made less unsightly by tattooing the opacity to resemble the normal pupil Even in eyes with some vision this procedure is often indicated and produces gratifying cosmetic results

Extraction of the Senile Cataract—Extracapsular extraction of a cataract (p 159) con

sists in opening the capsule of the cataractous lens and expressing its contents leavin the capsule in the eye The *intra capsular method* removes capsule and lens at one time The *extracapsular method* is somewhat easier and safer but an after cataract may form requiring *discission (needling)* at a later date

A successful intracapsular extraction gives a clear black pupil and no discission is required its major drawbacks are greater danger loss of vitreous and resultant chroni uveitis which may terminate in ultimate loss of the eye Either method in expert hands gives highly gratifying results in the large majority of instances The operations are done under local anesthesia

Intra and extracapsular extractions are preceded by careful preoperative surveys especially for focal infection oral sepsis diabetes and hypertension The patient remains in the hospital ten to fourteen days after operation Often a preliminary *iridectomy removing a* portion of the iris to form a keyhole pupil is performed After this operation the patient stays in the hospital for five days and returns two months later for the cataract extraction

Preliminary iridectomy is particularly advisable in patients with *hypertension* or *diabetes* In the pre- and postoperative care of the diabetic patient most ophthalmic surgeons limit the diet drastically rather than us insulin which they feel increases the tendency toward intra ocular hemorrhage

Discission of Secondary (After) Cataracts—If a membrane occurs following a cataract extraction and interferes with vision it may b incised or needled so as to effect a clear central opening This procedure requires only one or two days hospitalization Sometimes a more extensive procedure is necessary if the membrane is thick and tough

Discission of Congenital Cataracts—Discission of the congenital cataract (p 1594) requires that the capsule be incised in a number of places to allow free ingress of aqueous In time the soft cataract completely dissolves but a membrane may remain and demand a second discission The discission procedure is performed also for traumatic cataract in persons under the ages of twenty five to thirty years After that time the lens contains a nucleus which will not dissolve

Removal of Intra Ocular Foreign Bodies—A metallic intra ocular foreign body (p 1572) may be removed by a strong *magnet* applied to the eye An attempt is made to draw the substance into the anterior chamber from which it is removed after incision by means of a smaller magnet If the foreign body is nonmagnetic the problem is more difficult if an incision is made beyond and behind the lens considerable vitreous may be lost with the later danger of retinal detachment and loss of the eye by infection If magnetic bodies such as steel are not removed the eyeball will eventually degenerate (*siderosis bulbi*) Any intra ocular foreign body poses the hazard of *sympathetic ophthalmia* (p 1569)

Iridectomy for Acute Glaucoma—In acute glaucoma (p 1581) an incision is made into the eye in the angle of the anterior chamber 1 to 2 mm behind the limbus and a broad basal iridectomy is performed A good piece of iris is abscised at its root to facilitate drainage between anterior and posterior chambers The resultant defect in the iris gives the appearance of an inverted keyhole This operation is generally successful and when properly performed usually relieves the condition permanently

General anesthesia is sometimes preferred because the acute congestion of the eye results in poor absorption of the locally applied anesthetic

The Elliott Trephine Operation—The Elliot trephine operation is most commonly used for *chronic simple glaucoma* (p 1578) A small opening is made at the limbus to effect a permanent drainage between anterior and posterior chambers and conjunctiva This is an eminently successful maneuver and the eye continues to function well for a number of years Sometimes however a conjunctival bleb covering the trephine opening becomes edematous and infected Until the advent of sulfonamides this complication resulted in loss of the eye With modern chemotherapy the trephine bleb infection can be successfully combated

Lagrange Operation—The Lagrange operation is done for *secondary glaucoma* (p 1581) A portion of *sclera* is removed in addition to a basal *iridectomy*

Cyclodialysis—Cyclodialysis is often performed for *glaucoma* following cataract extraction (p 1581) The ciliary body is separated from the adjacent sclera with little trauma to the eye

Retinal Detachment Operations—The separated retina (p 1573) rarely reattaches spontaneously Until recently retinal detachments usually resulted in degeneration of the eye and secondary cataract formation

Present operative procedures which result in successful reattachment in at least 60 per

ment of patients consist of locating the retinal tear and surrounding it by a barrage of *diathermy punctures* in order to set up an *adhesive choroiditis* Postoperative care is more of an ordeal than the operation itself Both eyes must be bandaged to avoid movement the patient must remain flat in bed for at least two weeks Even after the dressings are removed glasses are worn to permit vision only through small central perforations

Operations on the Extra Ocular Muscles—Operative procedures are devised to *strengthen a muscle* by cutting off a piece reattaching the shortened muscle to its original insertion or advancing forward the insertion To *weaken a muscle* it is recessed the insertion is placed somewhat behind the original position or it is tenotomized or cut off from its insertion The last method may result in an overcorrection

Successful muscle operations depend on careful preoperative study and clean operative technic to prevent the formation of adhesions which may nullify the procedure The patient is warned in advance that a second operation may be necessary as it is far wiser to do a little less than to undertaken too much and overcorrect the condition

In children general anesthesia is usually employed but in adults local infiltration is preferable The operated eye is bandaged for four or five days and good cosmetic results are usually achieved

Enucleation of the Eye—The removal of an eye is a simple but harrowing procedure The implantation of a gold glass or plastic ball inside Tenon's capsule requires most careful surgery The operation is done under local anesthesia and the patient is not required to stay more than a day or to in the hospital unless an implant is performed

Exenteration of the Orbit—Exenteration of the orbit is required for *malignant orbital tumors* (p 1566) This very extensive procedure is not complete without the removal of everything in the orbit including periosteum

CHAPTER 76

THE EYE CONGENITAL ABNORMALITIES ANALOGUES OF THE DERMATOSES CYSTS AND NEOPLASMS

CONGENITAL ABNORMALITIES

Congenital abnormalities of the eye are usually clearly visible by direct inspection or by ophthalmoscopy (p 3628) Their recognition requires the practitioner to summon the specialist since certain of the anomalies are amenable to correction by methods of plastic surgery

CONGENITAL ANOMALIES OBSERVED BY INSPECTION

Albinism
Congenital absence of pigment Brows and lashes white Conjunctiva hyperemic Iris pale Pupil red Orange red fundus Optic disk almost indistinguishable High myopia Nystagmus Poor vision

Anophthalmos
Absence of eye due to failure of primary optic vesicle to bud from the cerebral vesicle

Blue Sclerotics
Associated with *fragilitas ossium* (osteogenesis imperfecta) multiple fractures and deafness (p 2879)

Buphthalmos
Ox like enlargement of globe due to obstruction to drainage of intra ocular fluid Intra ocular tension rises with *congenital glaucoma* Vision subnormal and optic atrophy may occur

Coloboma (Fetal Fissure)
Keyhole appearance of iris Defect of lens Pale white area due to defect of choroid and retina with a border of black pigment Branches of retinal vessels run across coloboma If optic nerve is involved large excavation appears usually on inferior aspect

Coloboma of Lids
Triangular notch of lid margins with absence of lashes and glands

Congenital Miosis
Absence of dilator muscle

Congenital Mydriasis
Absence of sphincter muscle

Corectopia
Anomalies in position of pupil

Corneal Opacities
Often associated with anterior synechia

Cyclops
Single eye in middle of forehead

Dermoid (p 1566)
At limbus in cornea or on conjunctiva Yellow flat oval tumor May contain hairs

Dermolipomas
Small yellow tumors in deep conjunctival tissues usually between superior and external recti

Distichiasis (p 1569)
Meibomian glands replaced by second row of incurving lashes



Fig 297—Congenital coloboma of eyelid



Fig 298—Epicanthus

Dyscoria
Slit shaped pupil as in cat

Embryotoxon
Annular opacity of periphery of cornea Continuous with sclera as contrasted with arcus senilis which has outer clear ring of cornea

Epicanthus
Bilateral skin fold projects over and partially covers inner angle of eye Normal in Mongolians

Epitarsus
Apron like fold of conjunctiva attached to inner tarsal surface of lid May be associated with congenital ectropion

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CHAPTER 76

THE EYE CONGENITAL ABNORMALITIES ANALOGUES OF THE DERMATOSES CYSTS AND NEOPLASMS

CONGENITAL ABNORMALITIES

CONGENITAL abnormalities of the eye are usually clearly visible by direct inspection or by ophthalmoscopy (p 3628) Their recognition requires the practitioner to summon the specialist since certain of the anomalies are amenable to correction by methods of plastic surgery

CONGENITAL ANOMALIES OBSERVED BY INSPECTION

Albinism

Congenital absence of pigment Brows and lashes white Conjunctiva hyperemic Iris pale Pupil red Orange red fundus Optic disk almost indistinguishable High myopia Nystagmus Poor vision

Anophthalmos

Absence of eye due to failure of primary optic vesicle to bud from the cerebral vesicle

Blue Sclerotics

Associated with *fragilitas ossium* (osteogenesis imperfecta) multiple fractures and deafness (p 2879)

Buphthalmos

Ox like enlargement of globe due to obstruction to drainage of intra ocular fluid Intra ocular tension rises with *congenital glaucoma* Vision subnormal and optic atrophy may occur

Coloboma (Fetal Fissure)

Keyhole appearance of iris Defect of lens Pale white area due to defect of choroid and retina with a border of black pigment Branches of retinal vessels run across coloboma If optic nerve is involved large excavation appears usually on inferior aspect

Coloboma of Lids

Triangular notch of lid margins with absence of lashes and glands

Congenital Miosis

Absence of dilator muscle

Congenital Mydriasis

Absence of sphincter muscle

Corectopia

Anomalies in position of pupil

Corneal Opacities

Often associated with anterior synechia

Conus or Inferior Crescent

Crescentic white area usually situated just beneath an elliptic distorted nerve head Condition non progressive as differentiated from myopic conus which is situated temporally

Cysts of Retina

Rare rounded tumors

Detachment of Retina

Rare anomaly (p 1573)

Ectopia Lentis

In association with arachnodactyly (elongation of bones of hands and feet) in Marfan s syndrome

Lenticonus

Elongated lens and visual distortion

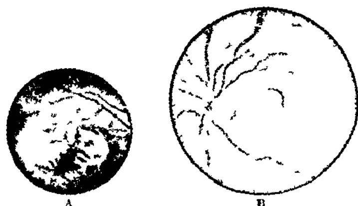


Fig 300 — A Coloboma of the macula B Medullated nerve fibers

Macular Coloboma

Large horizontal oval or round defect in macular region Stretched sclera seen on floor with black pigment Retinal vessels pass over area Considerable visual defect and nystagmus

Medullated Nerve Fibers

Striated white patch in superficial retinal layers covering the vessels in whole or part Characteristic frayed and feathered edge resembling shaving brush Partial scotoma may be caused by opaque character of medullated sheaths but usually no visual impairment

Melanosis Bulbi

Increase of pigment of uveal tract sclera iris and/or fundus

Melanosis of Retina

Grouped pigmented areas scattered through retina

Microphakia

Small lens

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Heterochromia

Color of irides differs

Megalocornea

Bilateral *enlargement of anterior segment of eye Large refractive error*

Microcornea

Cornea abnormally small in an otherwise normal eye Vision may be normal Tendency to glaucoma

Microphthalmos

Small eye which may be normal Usually associated with maldevelopment of retina and impaired vision Glaucoma often present

Polycoria

Two or more pupillary openings



Fig 299—Congenital ptosis in mother and son associated with almost complete external ophthalmoplegia *

Ptosis (p 1649)

Drooping of upper lid May be unilateral or bilateral

Stenosis of Nasolacrimal Ducts (p 1569)

Tearing and chronic dacryocystitis in infants

CONGENITAL ANOMALIES OBSERVED BY OPHTHALMOSCOPY

Aphakia

Absence of lens

Cataract

See p 1592

Choroideremia

Absence of choroid and pigment epithelium of retina Red reflex only in macula while rest of fundus is gleaming white Night blindness and concentric contraction of visual fields but normal color perception

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Oguchi s Disease
Congenital night blindness in Japanese

Persistence of Hyaloid Artery
Fine green gray cord extending from nerve head through central vitreous to posterior aspect of lens May have fine glial mantle May be complete or partial

Persistent Pupillary Membrane
Complete or partial remains of anterior part of vascular sheath of lens No interference with vision

Pigmentation of Optic Nerve
Spotty or uniform deposits

Pseudoneuritis
Heaping up of nerve fibers on optic disk with indistinctiveness of margins of disk and elevation of the nerve head resembling optic neuritis and papilledema Differentiate from *pathological* conditions by the fact that it is non progressive no hemorrhages or exudates are present arteries are not narrowed and veins are not engorged Usually but not invariably found in hyperopic eyes

Retinal Folds
Tent like ridges of retina project into vitreous Stretch from infero temporal portion of nerve head to periphery of retina May end at posterior aspect of lens producing cataractous change

Treatment —Confronted with a congenital anomaly of the visual organs it is the duty of the practitioner to refer the patient to the consultant ophthalmologist for expert opinion concerning prognosis and therapeutic possibilities Prophylactic measures against glaucoma (p 1578) are of value in *microphthalmos* and *buphthalmos* minor surgical procedures may be useful in *epitarsus* and *epicanthus* *dermoids* and *dermolipomas* are re moved for histological study the incurving lashes in a *distichiasis* should be epilated *stenosis of the nasolacrimal ducts* may respond to dilatation (p 1569)

ANALOGUES OF THE DERMATOSES

The skin of the eyelids often participates in the dermatoses Addition ally there may be involvement of conjunctiva cornea and sclera in cer tain of the more serious involvements of the skin

Acanthosis Nigricans (p 3357)
Conjunctivitis with papillary hypertrophy

Acne Rosacea (p 3357)
Blepharo conjunctivitis keratitis and episcleritis

Contact Dermatitis (Eczema) (p 3330)
Local drugs such as sulfonamides atropine anesthetics and mercury *Cosmetics* such as mascara nail polish nail dyes hair washes and rinses dyes rouge powder and lipstick

Dermatitis Herpetiformis (p 3371)
Conjunctival vesicles erosions xerosis keratinization and symblepharon (p 1569)

Epidermolysis Bullosa (p 3151)
Conjunctival vesicles corneal scarring ulcer and perforation



Fig 301—Atropine dermatitis



Fig 302—Pemphigus of conjunctiva with symblepharon

Erythema Multiforme (p 3374)
Catarrhal or purulent conjunctivitis corneal ulcers with perforations and phthisis bulbi associated with stomatitis and rash as *Stevens disease*

Hydroa (p 3176)
Severe conjunctivitis and keratitis

Ichthyosis (p 3152)
Chronic catarrhal conjunctivitis and keratitis

Lichen Planus (p 3389)
Rare involvement of conjunctiva and cornea

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CHAPTER 77

THE EYE INJURIES FROM MECHANICAL, PHYSICAL AND SYSTEMIC CAUSES

MECHANICAL DISTURBANCES

THE simpler mechanical disturbances of the eye are easy of recognition They require prompt reference to the specialist for the initiation of technical forms of therapy mostly in the nature of plastic surgery The required procedures often are simple of execution The risks of operation are minimal and the clinical results are highly satisfactory from the standpoint of cosmetic appearance and the prevention of ocular complications

Treatment—*The majority of mechanical disturbances require reference* to the specialist Plastic surgery may be of great value in ankyloblepharon blepharophimosis entropion eversion of the lacrimal punctum ptosis trichiasis and symblepharon Lagophthalmos may be improved by a tarsorrhaphy Dilatation and irrigation of the canaliculus is sometimes of value in occlusion of the lacrimal punctum For deficient lacrimation large doses of vitamin A and the instillation of Ringer s solution may be tried *with surgical closure of the tear puncta if non operative treatment fails*

INJURIES

The eye is vulnerable to many types of injuries particularly in modern industrial life Many carry serious threat of permanent damage to the visual apparatus and the practitioner is required to seek specialist consultation on frequent occasions There are few human tragedies which approach the horror of *sympathetic ophthalmia* (p 1569) in which loss of the traumatized eye is followed by destruction of the seemingly uninjured organ

The following chart deals briefly with the common ophthalmological injuries It is *followed immediately by more lengthy discussions of sympathetic* ophthalmia and the more serious injuries and mechanical afflictions of the visual apparatus

Treatment—Except for the extremely superficial injuries trauma to the eye requires reference to the specialist The practitioner should confine his efforts to the determination of underlying pathology and to symptomatic treatment Films of the skull and orbit are indicated with all mechanical *injuries especially those accompanied by emphysema* indicating communication with nose or accessory nasal sinuses

Symptomatic treatment consists of the application of compresses and the use of a pressure dressing until the patient may be seen by the ophthalmologist In the presence of perforating injuries it may be a wise precaution to initiate antibiotic treatment (p 106) with oral doses of *sulfadiazine and/or intramuscular injections of penicillin*

TABLE 107—MECHANICAL DISTURBANCES OF THE EYE THEIR ETIOLOGY AND CLINICAL MANIFESTATIONS

Lesion	Etiology	Clinical Manifestations
Ankyloblepharon	After burns and ulcers	Adhesion of upper to lower lid
Blepharophimosis	*Chronic conjunctivitis (p [illegible])*	*Contraction of palpebral fissure at outer canthus*
Deficient lacrimation	Congenital absence of glands Occlusion of ducts of gland Trachoma Vitamin A deficiency (p 616) Paralysis of C N V and VII	Keratoconjunctivitis sicca [illegible] xerosis [illegible] Sjögren's [illegible] with dryness of pharynx and parotid swelling
Ectropion	*Spastic* in young *Paralytic* with weakness of orbicularis *Senile* with relaxation of skin *Cicatricial* after wounds operations burns and ulcers	Exposure of lacrimal punctum with tearing Redness of exposed membranes Denudation of cornea Keratitis and ulceration (p 16[illegible])
Entropion	*Spastic* in elderly *Cicatricial* after trachoma burns injuries or operations on lids	Trichiasis Corneal irritation Photophobia Corneal ulceration vascularization and opacities
Eversion of lacrimal punctum	Relaxed lids in senility ectropion *facial palsy* (p 1481) *conjunctivitis*	Persistent tearing
Lagophthalmos	Proptosis (p [illegible]) Shortening of lids from burns or operations With ectropion or facial palsy (p 1481) In moribund and comatose	Incomplete closure of palpebral fissure Exposure of cornea with keratitis lagophthalmos (p 16[illegible]) Corneal opacities and ulceration Corneal perforation and panophthalmitis (p 15[illegible])
Occlusion of lacrimal puncta	Congenital Acquired from foreign body eyelash streptothrix concretion adhesions after inflammation	Punctum swollen Canaliculus pouch Pressure produces thin pus which may contain streptothrix
Ptosis	Congenital or acquired (p 1649)	*Droop of upper lid Congenital* [illegible] bilateral oculomotor is unilateral
Trichiasis	Follows blepharitis injuries burns and trachoma (p 16[illegible])	Inverted lashes irritate cornea
Symblepharon	After wounds burns and ulcers	Adhesions between eyelid and eyeball

SYMPATHETIC OPHTHALMIA

In sympathetic ophthalmia serous or plastic inflammation of the uveal tract of one eye follows a similar inflammatory process in the other In most instances the lesion in the exciting eye is a traumatic *iridocyclitis* due to a perforating injury in the region of the ciliary body The ophthal

mia in the sympathizing eye arises no sooner than three weeks following the injury to the exciting eye and it may be delayed for several years

Not all perforating injuries involving the ciliary body result in sympathetic ophthalmia Unfortunately however there is no way of determining which will result in this tragic occurrence and which will not

Clinical Manifestations —Following injury to the exciting eye the sympathizing eye becomes irritable and exhibits photophobia lacrimation pain and dimness of vision Examination discloses the presence of iridocyclitis of the exciting eye The sympathizing eye at first reveals nothing but tenderness over the ciliary region later irritative symptoms develop progressively or intermittently Under best circumstances the sympathetic iridocyclitis entirely disappears but in less favorable instances inflammatory signs develop and these may terminate in blindness Objective manifestations of sympathetic iridocyclitis include circumcorneal injection the presence of punctate deposits upon the posterior surface of the cornea (k p) miosis and increased intraocular tension the iris becomes thickened its markings are obliterated and extensive posterior synechiae develop plastic exudate fills up the pupil and the anterior chamber becomes



Fig 304 —Senile ectropion



Fig 305 —Symblepharon with pseudopterygium result of lime burn *

shallow opacities are demonstrable in the vitreous and the lens becomes opaque there may be final detachment of the retina and shrinking and atrophy of the eyeball

The mechanism by which sympathetic ophthalmia progresses is unknown The hypotheses which have been advanced include spread of infection from the sheath of one optic nerve to the other irritation of ciliary nerves action of a toxin generated by bacteria metastases through the blood stream of pathogenic bacteria or an allergic phenomenon

Treatment —Failure to enucleate or remove the exciting eye may result in blindness of the sympathizing eye Hence prophylactic removal of the sightless injured eye is the recommended procedure Once a sympathetic ophthalmia has become established removal of the exciting eye has no value and may be contra indicated if it possesses even slight vision

The ophthalmologist is faced with a difficult responsibility since he has no definitive method by which he can determine whether sympathetic ophthalmia will develop and the extent of blindness that will ensue Since there is a breathing spell of three weeks between injury and onset of

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sympathetic ophthalmia the practitioner has ample opportunity for obtaining several expert opinions before final action is taken In the mean

TABLE 108 —INJURIES OF THE EYE THEIR ETIOLOGY AND CLINICAL MANIFESTATIONS

Lesion	Etiology	Clinical Manifestations
Birth injury to cornea	After forceps delivery	Rupture of Descemet s membrane may result in permanent opacities
Contusion of retina (commotio retinae)	Blunt injury to eyeball	Impairment of vision with central scotoma Localized retinal edema at macula Milky appearance May clear completely or leave pigmented scar with permanent visual loss
Ecchymosis of lids	Local injury Fracture of base of skull (p 1450)	Black eye
Emphysema of lids	Fracture of orbital walls Communication with nose or accessory sinuses	Crepitus
Erosion of cornea	Scratches by nail edge of paper or twigs	*Denuded area stains green with fluorescein* (p 1518)
Injuries to optic nerve	With skull fracture (p 1450)	Usually loss of vision and optic atrophy after 4 to 6 weeks
Insect bites of lids		Considerable edema
Perforating injuries	Any small sharp object propelled with sufficient velocity	Vision often impaired Intra-ocular tension low Site of perforation may not be apparent Usually prolapse of iris and disorganization of eye May terminate in phthisis bulbi or sympathetic ophthalmia (p 1569)
Rupture of choroid	Blunt injury to eyeball	Crescentic white and hemorrhagic area May be extensive and cause loss of vision
Traumatic conjunctivitis	From irritants in industry such as acid vapors liquid or dust particles aniline tar and metal *wood or stone particles*	Pain irritation tearing and photophobia
Traumatic iridocyclitis	Blunt injury to eyeball	Blood in anterior chamber Low tension May terminate in atrophy of eyeball after protracted low grade inflammation
Vitreous hemorrhage	Blunt injury to eyeball	Marked visual impairment Black reflex with ophthalmoscope Fundus details obscured
Wounds of lid and conjunctiva	Local injury	Laceration

time he may employ non operative therapy such as foreign protein injection (p 1552) the administration of large doses of salicylates and body

baths of ultraviolet ray (p 1550) During this waiting period it is his responsibility to explain the situation to patient and next of kin so that all may have assurance when the final decision has been reached that the best of human judgment has been exercised

FOREIGN BODIES

Foreign bodies may enter the conjunctival sac or penetrate the cornea Injury to deeper structures carries the threat of sympathetic ophthalmia

In the Conjunctiva —*Nonmetallic foreign bodies* that lie free in the con junctival sac are removed with a moist applicator A few drops of a local anesthetic solution are instilled the lid is everted and the irritant is lifted off with care to avoid a scratch of the cornea

Penetration of the conjunctiva by *metallic objects* or *burned ashes* requires reference to the specialist who may be required to accomplish removal with a *spud* (p 1555) or any sharp instrument

Conjunctivitis nodosa results when the irritating hairs of caterpillars or plants become lodged in the conjunctival sac After removal frequent irrigations with saline solution are required

Corneal Foreign Bodies —Corneal foreign bodies require immediate re moval The resultant abrasion is best protected by instillations of castor oil and application of a protective dressing Penetration of the cornea places the uveal tract in jeopardy with the additional hazard of a *sympa thetic ophthalmia* (p 1569) Prophylactic anti infective therapy with sul fonamide and/or penicillin is advisable in the interim between injury and consultation with the ophthalmologist

BURNS

Eyelid conjunctiva and cornea may be exposed to burns by heat and chemicals Photoretinitis may follow injudicious exposure to brilliant sun light the glare of snow klieg lights sources of ultraviolet radiation and the flashes that result from welding and arc lights

Heat Burns —Burns of eyelids conjunctiva and cornea may result from steam gas ashes hot powder metals acids alkalies lime and irritant chemicals The immediate result is destruction of tissue such as that seen with burns elsewhere on the body In the conjunctiva the affected spot appears grey or white and is surrounded by redness and swelling of non escharotic membrane Corneal burns are associated with ulceration and necrosis

Treatment—Local anesthetization facilitates complete removal of for eign particles and residual chemicals This is particularly emphasized in lime burns since the offending substance may lodge under the upper lid and in the fornix with continuing trauma Burns that involve *lids* alone are relieved by local applications of castor oil and cold compresses of saline solution or iced milk With *conjunctival burns* the pupil is dilated with 2 per cent homatropine castor oil is instilled at frequent intervals cold compresses are applied and the lids are separated once daily using a glass rod anointed with petrolatum to prevent adhesions

Corneal burns are specialist province and are treated in the manner of conjunctival burns The eye must not be bandaged but is left open if the burn has resulted from exposure to lime or mustard gas Deep

corneal ulcerations may perforate and cause *panophthalmitis* (p 1571) with injury to the *uveal tract* and the hazard of *sympathetic ophthalmia* (p 1569)

The late treatment of conjunctival and corneal burns is concerned with the prevention and management of cicatricial deformities Healing may be followed by a *symblepharon entropion ectropion ankyloblepharon* and *lagophthalmos* (Table 107 p 569) Healing of the corneal ulcer may result in opacification with disturbances in vision Management of these *late sequels is specialist province*

Photoretinitis —Exposure to excessive sunlight the glare of snow Klieg and ultraviolet lights the flash of arc welding and the like may produce *conjunctivitis keratitis photoretinitis impairment of vision* or *central scotoma* These lesions are prevented by wearing tinted glasses (p 1558) or goggles and by the use of shields around the source of the ray After injury the patient requires isolation in a darkened room and application of cold compresses

DISPLACEMENT OF THE LENS

Dislocation or *luxation* of the lens and partial dislocation or subluxation may be congenital or acquired The disturbances are associated with defects in the suspensory ligament resulting from atrophy rupture stretching or imperfect development

The *congenital type* is generally partial tending to become complete in after years It is generally bilateral symmetrical and hereditary it may be associated with *arachnodactyly* (Marfan s disease) *Acquired dislocations* of the lens are traumatic or spontaneous In the traumatic type a direct blow upon the eye is the usual cause In spontaneous dislocation while the exciting cause may appear to be insignificant a defect in the zonule may result in lens displacement Ocular conditions which predispose to dislocation include *high myopia fluid vitreous choroiditis detachment of the retina* and *hypermature cataract*

Clinical Manifestations —The clinical manifestations that suggest dislocation of the lens include *visual disturbances impairment of accommodation changes in the refractive power* of the eye and *monocular diplopia* Partial dislocation or subluxation may be of minimal degree and may only be diagnosed by the observation of a tremulous condition of the iris (*iridodonesis*) Unequal depth of the anterior chamber may be noticed If subluxation is marked the border of the lens is visible in the pupillary area Complete dislocation may result in displacement of the lens anteriorly into aqueous or posteriorly into vitreous

Treatment —The treatment of lens dislocations lies in the hands of the specialist In partial dislocation it may be necessary only to prescribe proper glasses If the lens is dislocated into the anterior chamber the resulting inflammation with acute secondary glaucoma may require removal of the lens (p 1558) Dislocation into vitreous is ultimately more serious as removal of the lens then is technically more difficult

DETACHMENT OF THE RETINA

Detachment of the retina is a serious and not uncommon condition A hole or a tear in the retina resulting from retinal disease or injury is the main factor in the production and persistence of the detachment

Most detachments occur in people from fifty to sixty years of age and about two thirds are in myopes They generally occur after trauma often of minimal degree in persons predisposed because of senility and myopia Other causes include exudative chorioretinitis long standing iridocyclitis hypertensive retinopathies eclampsia and choroidal tumors and hemorrhages

DIFFERENTIAL DIAGNOSIS OF

Photophobia

Photophobia is usually accompanied by blepharospasm and may be caused by local and systemic disturbances

	DIAGNOSTIC FEATURES
Functional	Fatigue asthenopia neuroses and hysteria Order refraction (p 1537)
Adnexal	Foreign bodies local injuries physical and mechanical lesions of lids and conjunctivae Blepharitis Conjunctivitis Analogues of dermatoses Herpes ophthalmicus Make local examination with oblique illumination (p 3622) Identify pathogen by stains of exudat and epithelial scrapings (p 1546)
Lesions of Fibrous Tunic	Keratitis scleritis episcleritis or corneal ulcer Refer to specialist for slit lamp examination and epithelial scrapings (p 1546)
Lesions of Vascular Tunic	Iritis iridocyclitis or sympathetic ophthalmia Refer to specialist for examination by slit lamp and ophthalmoscope (p 1545)
Lesions of Neural Elements	Retinitis and retinitis pigmentosa Refer to specialist for fundus examination
Disturbances of Intra ocular Tension	Glaucoma with pain in eye elevation of tension and relief from miotics Avoid mydriatics
Systemic Infections	Upper respiratory infection with nasopharyngitis Measles with characteristic exanthem Pertussis with paroxysmal cough and lymphocytosis Meningitis and encephalitis with organic neurologic findings and changes in cerebrospinal fluid Variola varicella and rubeola with characteristic rashes (p 172) Poliomyelitis with evidences of anterior horn involvement (p 457)
Metabolic Disturbances and Poisonings	Migraine and epilepsy Allergic conjunctivitis and rhinitis with eosinophilia Vitamin A deficiency with night blindness (p 1535) Cinchonism (p 862)
Neurogenic	Facial and trigeminal neuralgias (p 1484)

Clinical Manifestations—In retinal detachment the patient first may notice *spots or a cloud before the eyes* and *flashes of light* (*photopsia*) If detachment involves the macula central vision is impaired

Ophthalmoscopy—Ophthalmoscopic examination usually reveals a fluid vitreous containing numerous filmy floating opacities The retina is thrown into folds appears considerably paler than normal and finally assumes a

greyish tint The detached retina is brought into focus with much stronger plus lenses than the remainder of the retina The retinal vessels appear black in contrast to the red vessels seen elsewhere they seem small and lose their central reflex The *retinal tear* usually is situated toward the periphery and appears as a bright red spot The color is due to choroid shining through the hole Successful operative treatment depends on the accurate localization of the tear

Course and Treatment—The great majority of *untreated retinal detachments* terminate in complete detachment degeneration of the eye and blindness Spontaneous reattachment rarely occurs

DIFFERENTIAL DIAGNOSIS OF

Exophthalmos (Proptosis)

Bulging of the eyeball may be unilateral or bilateral In either instance the etiologic factor may be local or systemic

	DIAGNOSTIC FEATURES
Ophthalmic	Edema inflammation or neoplasm of orbit with visible and palpable changes (p 1615) Cavernous sinus thrombosis with fever and bacteremia (p 1447) High myopia with fundus changes and refractive error (p 1536) Varicosities of orbit with increased bulging when head is lowered.
Lesions of Contiguous Structures	Hydrocephalus with pumpkin head. Suppurative spheno-ethmoiditis with radiographic changes and demonstrable pus
Metabolic	Hyperthyroidism with elevation of B M R and therapeutic response to iodide Hutchinson s tumor of the adrenals in infancy Schuller Hand Christian disease with diabetes insipidus and cholesterol deposits in subcutaneous tissues and bones Poisoning with potassium cyanide
Systemic Infection	Epidemic encephalitis with neurologic findings and changes in cerebrospinal fluid (p 441)
Vascular	Arteriovenous aneurysm of internal parotid and cavernous sinus with pulsating exophthalmos

Operative treatment (p 1558) represents a great advance in opthalmology and produces a satisfactory result in more than 60 per cent of patients Reoperation greatly increases favorable results With associated pre existent inflammation of retinopathy surgery is contraindicated

EXOPHTHALMOS (PROPTOSIS)

Exophthalmos is an undue protrusion of eye from orbit as measured by the exophthalmometer (p 1546) The condition most often results from the presence of *hyperthyroidism*

Clinical Manifestations—Marked exophthalmos causes *impaired motility* of the eyeball *diplopia* and *lagophthalmos* (p 1569) The latter may result in exposure of cornea and keratitis with ultimate loss of the eye if untreated Involvement of the optic nerve leads to impairment of vision and the presence of a neuritis

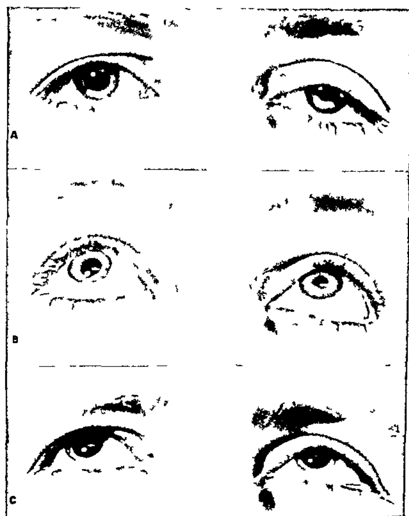


Fig 306—*A* Paralysis of the left sympathetic nerve Note the small palpebral fissure, the lowering of the upper eyelid (pseudoptosis) and small pupil also the slight enophthalmos *B* Picture taken one hour after several drops of 4 per cent cocaine had been instilled The pupil did not dilate and the palpebral fissure remained the same *C* Shows the effect of homatropine instilled in each eye Both pupils well dilated in twenty minutes

Treatment—Treatment is directed at the underlying cause The practitioner should warn the hyperthyroid patient that there may be little regression of the exophthalmos following iodine and deracil therapy or thyroidectomy (p 1214) In the presence of lagophthalmos plastic surgery of the lid may be required More formidable procedures include re-

* Casten in Berens The Eye and Its Diseases

moval of orbital fat ganglionectomy and the Naffziger operation upon the orbit

In hyperpituitary exophthalmos thyroidectomy may markedly aggravate the exophthalmos in such instances thyroid extract or iodine may be helpful plastic surgery of the lids is contraindicated and decompression of the orbit may be urgently required

PULSATING EXOPHTHALMOS

In pulsating exophthalmos a *bruit* is audible when the stethoscope is placed over eye or forehead The patient complains of head noises and local pain the observer notes marked dilatation of the vessels of the retina conjunctiva and lids Vision may be impaired and an optic neuritis may develop Not infrequently there are associated paralyses of third and sixth cranial nerves (p 1645 1647)

Compression of the internal carotid artery causes pulsations and bruit to diminish or disappear since the causative mechanism is usually an *arteriovenous aneurysm* involving internal carotid artery and cavernous sinus The condition may be relieved by gradual compression of the carotid first on one side and then if necessary on the other There is no necessity to institute therapy unless for cosmetic reasons in which case plastic surgery is recommended

ENOPHTHALMOS

Enophthalmos is rare It occurs with decrease in the amount of orbital fat with dehydration such as that occurring in cholera following paralysis of the upper sympathetic chain (Horner s syndrome) following injury to the margin of the orbit and after operations on the recti

PAPILLEDEMA

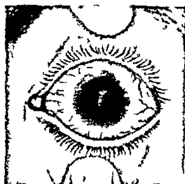
Pathogenesis—Pathologically papilledema is a simple swelling of the optic nerve head due to edema of the individual nerve fibers The accompanying venous congestion superimposes a metabolic disturbance which interferes with the normal flow of tissue fluid

Clinical Manifestations—Papilledema is not necessarily associated with derangement of visual acuity Only when the condition has been present for a considerable period of time does diminution of vision occur When using the ophthalmoscope the changes first noted are *increased redness of the disk* with slight *haziness and blurring of the upper and lower margins* especially on the nasal side There may be some *fullness of the veins* the surrounding retina shows greyish white striations as evidence of edema

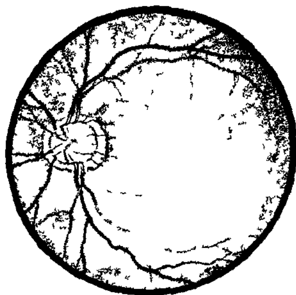
Once established the ophthalmoscopic appearance of papilledema is very marked There is considerable swelling of the disk with obvious blurring of the margin so that it is difficult to demarcate the nerve head from the edematous retina Sometimes only the confluence of the larger vessels indicates the position of the disk hemorrhages and exudates appear the veins become enormously distended and tortuous while the arteries remain small

The *amount of elevation* of the nerve head is measured by the specialist who notes the strongest plus lens with which he can see the vessels of the disk as compared to the strongest plus lens with which he can see

is grayish in color and there is *cupping* or *excavation* involving the central area with a peripheral overhanging edge Cupping is due to increased tension and may be appreciated by following the course of the *central retinal vessels* they are pushed toward the nasal side and climb over the sharp overhanging rim with an acute bend when tension is very high *arterial pulsations* may occur Cupping does not occur until glaucoma has been well established over a period of years



A



B

Fig 308—*A* Acute glaucoma *B* Cupping of the disk in chronic glaucoma

Elevation of Intra ocular Tension—The important diagnostic findi in simple glaucoma is *elevated intra ocular tension* which is present fro the onset although it may become higher during the course of the disea This may be ascertained by palpation of the eye with the fingers Tl test should be performed by practitioners whenever the slightest su picion of glaucoma exists If it appears that an increased tension is pre

* Gifford Textbook of Ophthalmology

ent the patient is sent to the specialist for *tonometric examination* (p 1545)

Course—Chronic simple glaucoma if unarrested eventually goes on to complete blindness

Treatment—The treatment of simple glaucoma belongs in the hands of the specialist who controls the tension by the use of *miotics* (p 105) The patient is observed periodically and special attention is directed to visual acuity and fields and intra-ocular tension If the condition progresses one of the type of drainage operations is advised (p 1558) The most usual procedure is the Elliot *trephine* operation (p 1558)

Congestive Glaucoma—Congestive glaucoma may be acute or chronic Acute congestive glaucoma may become superimposed upon simple glaucoma or it may appear without warning and with great suddenness in an eye that is apparently healthy It is usually preceded by mild *prodromal* attacks with diminution in acuity of vision fogginess and the appearance of a ring of rainbow tints around foci of light

Clinical Manifestations—In acute congestive glaucoma the patient is generally awakened by excruciating pain in the eye profuse *lacrimation* and intense *trigeminal neuralgia* He notes rapid *failure of vision* which may progress to loss of light perception within a few hours The local symptoms are associated with *nausea* and *vomiting* evidences of shock or flushing and a slight pyrexia

Intra ocular tension is markedly increased as revealed by comparing the palpatory sensations in the affected as against the nonaffected eye The involved eye is intensely red and congested The lids are edematous the conjunctiva chemotic the globe tender and the cornea is dull and steamy The pupil is widely dilated vertically oval in shape and unresponsive to light (p 1533) Ophthalmoscopic examination is difficult because of edema of the cornea and if the optic disk can be seen it is edematous and hyperemic

Course—Following the acute phase the eye may seem normal but actually it has progressed to the stage of *chronic congestive glaucoma* The symptoms and signs persist to a varying degree exacerbations of acute glaucoma are encountered and the final stage is that of *absolute glaucoma* resulting in a blinded hard and painful eye

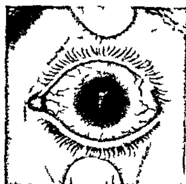
Treatment—In the acute phases cold compresses are applied and *opiates* are given in large doses A combination of 2 per cent *pilocarpine* and 0.5 per cent *eserine* is instilled into the eye every ten or fifteen minutes for 6 doses and then every two hours to effect complete miosis and reduction of intra ocular tension Meanwhile the specialist is consulted Within a few hours the miotics may result in a dramatic subsidence of symptoms and serve as a therapeutic test If relief is not obtained the ophthalmologist employs instillations of 0 1 to 0 2 diisopropyl fluorophosphate

The operation of choice is an *iridectomy* (p 1558) which should be performed even if the tension is reduced to normal A successful iridectomy usually results in permanent and complete relief of acute congestive glaucoma Less satisfactory results are observed in chronic simple glaucoma

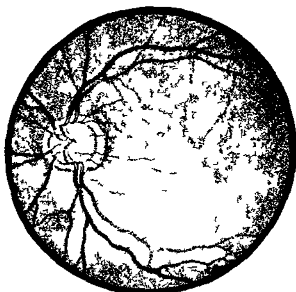
SECONDARY GLAUCOMA

In secondary glaucoma a recognized pathologic lesion of the eye is complicated by increase in intra ocular pressure As the basic condition

is grayish in color and there is *cupping* or *excavation* involving the central area with a peripheral overhanging edge Cupping is due to increased tension and may be appreciated by following the course of the *central retinal vessels* they are pushed toward the nasal side and climb over the sharp overhanging rim with an acute bend when tension is very high *arterial pulsations* may occur Cupping does not occur until glaucoma has been well established over a period of years



A



B

Fig 308—*A* Acute glaucoma *B* Cupping of the disk in chronic glaucoma

Elevation of Intra ocular Tension—The important diagnostic finding *in simple glaucoma is elevated intra ocular tension* which is present from the onset although it may become higher during the course of the disease This may be ascertained by palpation of the eye with the fingers This test should be performed by practitioners whenever the slightest suspicion of glaucoma exists If it appears that an increased tension is pres

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Etiology and Clinical Manifestations —The common basic lesions in secondary glaucoma include intra ocular inflammation trauma changes in the lens vascular anomalies of the retina intra ocular tumors detachment of the retina intra ocular hemorrhage atrophic and sclerotic conditions of the eye venous obstruction in the orbit and congenital anomalies The clinical manifestations are those of increased intra ocular tension (p 1526) superimposed on the more fundamental lesion

Treatment —The treatment of secondary glaucoma depends on the nature of the underlying condition If control is possible the glaucoma may disappear Otherwise *iridectomy* (p 1558) or the Lagrange operation (p 1558) is indicated

EPIDEMIC DROPSY

The condition which is seen in India and is known as *epidemic dropsy* is characterized by glaucoma (p 1578) general edema hypertrophy of the heart and a peripheral neuritis suggesting a thiamine deficiency (p 616) Medicinal treatment is of no avail and interestingly enough only operation is of help

OCULAR HYPOTENSION

Ocular hypotension or *ophthalmomalacia* occurs in a number of conditions such as myopia retinal detachment trauma infections and inflammations of the eye Hypotension may occur in severe general illnesses particularly poisonings by *quinine* or *barbiturates* in *diabetic coma* and in primary anemias

Although the clinical symptoms of hypotony are by no means striking grave visual disability may appear and persist The *treatment* of this condition requires attention to the elimination or correction of the more basic abnormality

DIFFERENTIAL DIAGNOSIS OF

Pain in the Eye

The complaint of pain in the eye requires intensive investigation At times the cause may be as simple as a cinder located in the conjunctival sac but again the disturbing circum tances may be a mechanism as serious as acute glaucoma

	DIAGNOSTIC FEATURES
Functional	Ametropia pre byopia asthenopia or pasm of accommodation Correct refractive error (p 1536)
Psychogenic	Malingering and hysteria Normal findings on examination
Neurogenic	Trigeminal neuralgias
Adnexal	Physical chemical and mechanical injuries to the eye Foreign body Exposure to tear-gas Hordeolum chalazion dacryocystitis or con junctivitis Examine by oblique illumination and inversion of lid (p 3622) Make cultures and smears from exudate and epithelial scrap ings (p 1546) Look for analogues of the der matoses particularly herpes ophthalmicus with characteristic vesicle (p 1629)
D sturbances of Fibrous Tunic	Keratitis episcleritis scleritis and corneal ulcer Refer to peciali t for slit lamp examina tion and for microscopy of epithelial scrapings (p 1546)
Disturbances of Vascular Tunic	Choroiditis iridocyclitis sympathetic oph thalmia Refer to specialist for slit lamp and fundus examinations (p 1545)
Disturbances of Neural Elements	Retinitis photoretinitis and obstructions of retinal arteries or veins Cavernous sinus *thrombosis Refer to pecialist for fundus ex* amination (p 1545)
Disturbances of Intra ocular Tension	Glaucoma with hard eyeball and relief from use of miotics Avoid mydriatics
Orbital	Orbital cellulitis periostitis and osteomyelitis Get x rays of skull and nasal accessory sinuses Consult rhinologist and ophthalmologist
Contiguous Infections	Particularly acute inflammations of nasal accessory sinuses Get x rays and consult rhinologist for local examination and lavage of sphenoids
Systemic Infections	Upper respiratory infection with nasopharyn gitis Measles with cutaneous eruption Dengue in epidemic areas Influenza with respiratory manifestations and myalgias Men ingococcemia with bacteremia and general ized eruption
Metabolic	Particularly migraine and epilepsy

has usually brought the patient to the specialist and the glaucoma gener ally develops during treatment the problem is not so urgent a concern of the practitioner as are the primary varieties

General Paresis (p 1377)
Argyll Robertson pupil Ocular palsy Optic neuritis and atrophy

Hepato lenticular Degeneration (p 1418 1977)
Kayser Fleischer ring of greenish brown pigmentation in deep layers of cornea

Hysteria (p 1353)
Impairment of vision amaurosis perversions of color vision micropsia macropsia nyctalopia diplopia monocular diplopia anesthetic cornea photophobia disturbances of intra and extra ocular muscles and tubular field defects (gun barrel)

Increased Intracranial Pressure (p 1421)
Papilledema

Meningitis (p 1462)
Oculomotor paralysis Optic neuritis

Mongolian Idiocy (p 2774)
Slit eyes and lenticular opacities

Multiple Sclerosis (p 1504)
Retrobulbar neuritis central scotoma nystagmus transient oculomotor paralyses and temporal pallor of disk

Myasthenia Gravis (p 2886)
Ptosis oculomotor paralyses diplopia and impaired convergence and accommodation

Myotonia Atrophica (p 2888)
Cataract

Myotonia Congenita (p 2886)
Cataract

Optic Pathway Lesion (p 1645)
Homonymous hemianopsia

Occipital Lobe Lesion (p 1426)
Localized field defects or perceptual blindness and papilledema

Pressure on Optic Nerve (p 1645)
Blindness loss of pupillary reflex and scotomas

Quadrigeminal Plate Lesion (p 1426)
Paralysis of upward gaze diplopia Argyll Robertson pupils and papilledema

Red Nucleus Lesion (p 1426)
Bilateral papilledema with homolateral oculomotor paralysis

Schilder s Disease (p 1640)
Optic neuritis and bilateral atrophy

Tabes Dorsalis (p 1464)
As general paresis

CHAPTER 78

OPHTHALMIC MANIFESTATIONS OF NEUROMUSCULAR, CARDIOVASCULAR AND HEMATOPOIETIC DISORDERS

LOCAL ocular manifestations are frequently encountered in disturbances of the neuromuscular cardiovascular and hematopoietic systems These phenomena are easily visible herald signs of profound afflictions which otherwise may be unsuspected The recognition of the causal association has therapeutic as well as diagnostic significance since indication is afforded for a plan of treatment of wider scope than mere local or symptomatic measures

OPHTHALMIC MANIFESTATIONS OF NEUROMUSCULAR DISORDERS

Because of the clarity with which the optic nerves may be viewed by ophthalmoscopy neurologic disturbances are often first recognized by the manifestations which are here encountered Other aids to neurologic diagnosis are afforded by disturbances of the visual field and of the extraocular muscles that are innervated by the cranial nerves

Amaurotic Family Idiocy (p 1584)
Optic atrophy with cherry red spot at macula

Central Chiasm Lesion (p 1645)
Bitemporal hemianopsia and optic atrophy

Cerebellar Lesion (p 1426)
Bilateral papilledema Bilateral partial oculomotor paralysis

Cerebellopontine Angle Lesion (p 1426)
Bilateral papilledema Homolateral anesthesia of cornea Homolateral abducens paralyses and nystagmus

Chorea (p 191)
Blepharospasm

Encephalitis (p 441)
Ocular palsies and the syndrome of paralysis agitans

Epilepsy and Migraine (p 1506 1515)
Scintillating scotomas and ophthalmoplegia

Friedreich s Ataxia (p 1415)
Pseudonystagmus

Frontal Lobe Tumor (p 1425)
Foster Kennedy syndrome of ipsilateral optic atrophy and contralateral papilledema

Cerebral Aneurysms (p 968)
Oculomotor paralyses

Malignant Hypertension
Arteriosclerotic changes plus papilledema Venous engorgement retinal edema and retinal detachment and cotton wool exudates Radiating star figures at macular Flame shaped hemorrhages

Pulmonary Stenosis (p 971)
Congestion tortuosity and cyanosis of retinal vessels

Raynaud's Disease (p 1000)
Spasm of retinal vessels

Subacute Bacterial Endocarditis (p 1021)
Conjunctival and retinal petechiae Superficial retinal hemorrhages Embolization of central artery

Thrombo angiitis Obliterans (p 1029)
Vessels appear as white strands due to peri and endovasculitis

Thrombosis of Cavernous Sinus
Immobility of eye with increasing exophthalmos Distention of retinal veins papilledema and optic neuritis Edema over mastoid

Varicose Veins of Orbit
Intermittent exophthalmos which lessens when head is held erect and increases when head is depressed

OBSTRUCTION OF THE CENTRAL RETINAL ARTERY

Obstruction of the central retinal artery may be due to spasm or organic closure Spastic occlusion occurs with systemic evidences of vasomotor instability particularly in migraine allergy and hypertension Organic blockage is most often due to arterial thrombosis associated with a generalized endarteritis but it may also follow embolism in cardiac disease or subacute bacterial endocarditis

Clinical Manifestations —*Obstruction of the central retinal artery is an* ophthalmic emergency in which the patient becomes suddenly and almost completely blinded The left eye is more generally affected in an embolization and the affliction is characterized by the fact that it is not accompanied by pain When the main vessel is occluded blindness is complete even to the perception of light If a branch is blocked there may be only a sector shaped defect of the visual field

Ophthalmoscopy —The ophthalmoscopic picture of complete closure of the central retinal artery is very characteristic The abnormally pale nerve head is obscured by edema The arteries are thinned and can be followed for only a short distance from the disk The veins are also narrowed and the blood column may be broken up into segments giving a beaded or fragmented appearance In a short time the retina loses its transparency and becomes pale edematous and milky Later at the macula a bright cherry red spot is observed where the red reflex of the choroid shines through the thin fovea standing out in marked contrast to the neighboring whitish retina If a collateral cilioretinal artery is present an island of

Temporal Lobe Lesion (p 1426)
Papilledema and hemianopic field defect

Trigeminal Neuralgia (p 1482)
Pain in eye

OPHTHALMIC MANIFESTATIONS OF CARDIOVASCULAR DISEASE

The ophthalmic manifestations of cardiovascular disease for the most part are local manifestations of a systemic disturbance Only in rare instances such as obstruction of the central retinal artery or veins is the clinical condition a predominantly isolated affliction

Acute Glomerulonephritis
See Hypertensive Retinopathy p 2373

Aortic Aneurysm (p 1026)
Mydriasis and widening of palpebral fissure

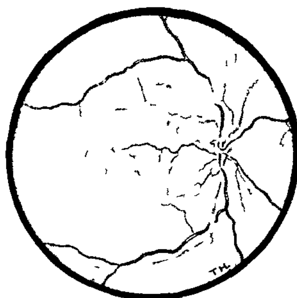


Fig 309 —Arteriosclerotic retinopathy

Aortic Insufficiency (p 970)
Arterial pulsations of retinal artery due to high pulse pressure

Arteriosclerosis
Increased translucency of arteries (copper wire and silver wire) Beading tortuosity and attenuation of arteries and sheathing of vessels due to fibrous perivasculitis Constriction of veins (Gunn s sign) at arteriovenous crossing Irregularity of vascular lumen Thrombosis of central artery or vein Hemorrhages and exudates

Arteriovenous Aneurysm of Internal Carotid and Cavernous Sinus
Pulsating exophthalmos (p 1577) with bruit Dilatation of vessels of retina conjunctiva and lids Optic neuritis Pulsation lessened by compression of internal carotid

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Treatment.—Whatever the cause of [illegible] artery prompt treatment may save [illegible] Vaso[illegible] by inhalations of amyl nitrite [illegible] dermic injection of acetylcholine or the intravenous [illegible] or aminophyllin. The globe may be massaged and [illegible] drate a patient [illegible] in order to lower its intraocular pressure. [illegible] any of the various forms of therapy [illegible] improvement may occur. Nevertheless, the [illegible] too great reliance upon the healing [illegible] of knowing which of the [illegible] may be permanent.

OBSTRUCTION OF THE RETINAL VEINS

Obstruction of the retinal vein or one of its branches is [illegible] than closure of an artery. Whereas in the occlusion of an artery the worst complication is blindness in that eye, closure of the central retinal



Fig 311 —Pernicious anemia[1]

vein may be associated with *secondary glaucoma* (p 1581) and terrific pain amenable to no therapy save removal of the eye. The condition usually occurs in older people when it is associated with arteriosclerosis, slowing of the blood flow and endophlebitis. In young people it may follow febrile diseases such as influenza.

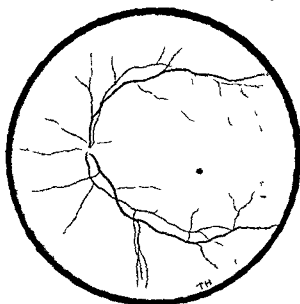
Clinical Manifestations.—At the time of the accident the symptoms are confined to *loss of vision*. In complete thrombosis loss of sight comes on rapidly but not with the dramatic suddenness and completeness of arterial obstruction. In thrombosis of one of the branches of the central retinal veins the loss of vision is limited to the area served by the occluded branch.

Ophthalmoscopy.—When the trunk of the central vein is blocked the ophthalmoscopic picture is very striking, the nerve head is completely obscured by edema and numerous *linear hemorrhages* are arranged radially. Scattered throughout the rest of the fundus are other hemorrhages.

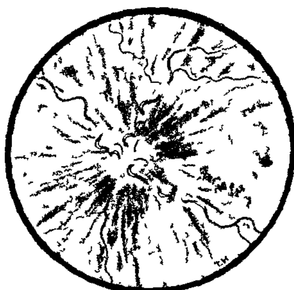
[1] Courtesy of Dr Arthur Bedell

relatively normal retina may remain in the central area with preservation of central vision

If obstruction of the artery is not overcome the retina atrophies after a few days and later there is optic atrophy The end picture is character



A



B

Fig 310—*A* Closure of central retinal artery *B* Thrombosis of central retinal vein

ized by the appearance of thin shrunken arteries represented by white lines atrophy of the nerve fibers and the ganglion cell layers of the retina with preservation of the outer retinal layers

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CHAPTER 79

OPHTHALMIC DISTURBANCES IN METABOLIC DISORDERS AND POISONINGS

METABOLIC disorders and poisonings may be reflected in the visual apparatus by a variety of manifestations The most frequent of these is the cataract later described in detail (p 1592) The less common are tabulated in *Table 109*

TABLE 109—OPHTHALMIC MANIFESTATIONS OF METABOLIC DISORDERS AND POISONINGS

Lesion	Site	Associated Condition
Pinguecula	Yellow triangular patches situated in bulbar conjunctiva on either side of cornea	Following exposure and in senility Brown pinguecula in Gaucher's disease (p 1133)
Xerosis	Of the conjunctiva	With burns trachoma or vitamin A deficiency
Pigmentation	Of sclera	Ecchymosis following subconjunctival hemorrhage Yellow tint in icterus and carotinemia Black pigmentation in Addison's disease and ochronosis Blue sclerotics in fragilitas ossium Silver deposits in argyrosis Kayser Fleischer ring in hepatolenticular degeneration (pp 1418 19)
Arcus senilis (gerontoxon)	Opaque greyish white ring just within corneal margin	Familial In arteriosclerosis
Embryotoxon	At margin of cornea continuous with sclera	Congenital anomaly
Keratomalacia	Of cornea	In vitamin A deficiency
Dystrophies	Fine opacities and vacuoles in *endothelium and epithelium of cornea*	May be familial and hereditary or *related to arteriosclerosis*
Drusen (colloid) bodies	Of choroid appearing as hyaline excrescence of lamina vitrea	Degenerative phenomena
Angioid streaks	Reddish black brown lines of choroid situated around the disk	With pseudoxanthoma elasticum
Macular degeneration	Small hemorrhages of retina followed by pigmentary or cystic changes May produce hole in macula with the appearance of a deep-red punched-out area	Usually senile

of various types and sizes The fundus picture is dominated by the enormously distended and *tortuous retinal veins* which may be several times their normal size The arteries are narrowed

In a branch thrombosis the pathology is limited to the area supplied by the tributary veins The condition is readily diagnosed by the enormous dilatation of the occluded branch as compared to other uninvolved branches Perhaps even more characteristic are the numerous hemorrhages which radiate out from the nerve head forming a triangle with apex adjacent to nerve head

Treatment—*Heparinization* (p 1050) by the Loewe method or the oral use of dicoumarin (p 1050) should be undertaken without even waiting the arrival of the consultant ophthalmologist The complication of secondary glaucoma impends in 10 to 20 per cent of instances when the central retinal vein is occluded In *branch thrombosis* glaucoma does not occur Needless to say mydriatics are contraindicated under any circumstances

OPHTHALMIC MANIFFSTATIONS OF HEMATOPOIETIC DISORDERS

Diseases of the blood and blood forming organs are prone to be reflected in the delicate tissues of the organs of vision The local manifestations include hemorrhage edema and thromboses

Anemia of Hemorrhage (p 1056)
Acute edema of optic nerve and retina may cause transient blindness

Hyperchromic Anemia (p 1077)
Pallor of nerve narrow and pale retinal arteries flame shaped superficial hemorrhages and cotton like exudates

Hypochromic Anemia (p 1089)
If severe may cause retinal hemorrhages and optic neuritis Pale conjunctiva Pearly sclera

Leukemias (p 1100)
May be associated with edema of disk exudates in retina and white centered hemorrhages

Polycythemia (p 1092)
May result in distention and engorgement of veins purple color to retina and congestion of conjunctiva

Purpuras (p 1121)
May cause subconjunctival retinal hemorrhages edema and neuritis of optic nerve

Hemophilia (p 1118)
May produce hyphemia and hemorrhages of retina and orbit

no clinical significance and complete cataracts involve almost the whole lens

If vision is impaired to any significant extent *treatment* is in the hands of the specialist who usually performs a *discission* (p 1558) The retina and macula often do not develop perfectly in the presence of congenital cataract and even after an eminently successful operation vision may be considerably impaired

Degenerative Cataract —The *senile* degenerative cataract is the most frequent clinical type It is quite common after the fiftieth year although sometimes it is seen as early as forty Both eyes usually are involved but



Fig 312 —Arcus senilis

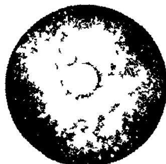


Fig 313 —Cholesterol bodies in the vitreous (synchysis scintillans)



Fig 314 —Mature senile cataract

generally one is more affected than the other The development of the cataract is variable growth may be slow or rapid In the incipient stage of a senile cataract the opacity is usually characterized by streaklike spots As the lens absorbs fluid and swells the cataract enters the *immature stage* When the lens loses most of its fluid and shrinks it enters the *mature stage* and becomes opaque and of a dull grey or amber color The stage of *hypermaturity* may be associated with dislocation of the lens and degenerative changes that terminate in loss of the eye

Pathologic Cataract —The pathological cataract may be associated with a variety of local and systemic disturbances such as osmotic changes in

TABLE 109.—OPHTHALMIC MANIFESTATIONS OF METABOLIC DISORDERS AND POISONINGS (*Continued*)

Lesion	Site	Associated Condition
Retinitis pigmentosa	A hereditary chronic and bilateral process beginning in childhood and terminating in blindness by middle life A degeneration of retinal neuro-epithelium particularly of the rods Results in night blindness increasing contraction of peripheral fields with eventual impairment of central vision Ophthalmoscopy reveals attenuation of retinal vessels pigmentary deposits and a yellow waxy appearance of the atrophic disk	Of unknown origin but may be complicated by cataract and glaucoma.
Macular and lipoid degeneration	Of retina	In amaurotic family idiocy lipoid histiocytosis (Niemann Pick) and Laurence-Moon Biedl syndrome
Dust like haziness	Of vitreous	In cyclitis and choroiditis
Asteroid bodies	Snow ball opacity in vitreous	In advanced age
Synchysis scintillans	Showers of moving silvery and golden particles of cholesterol in degenerated and fluid vitreous	Of unknown etiology
Muscae volitantes	Subjective spots before the eyes"	With errors of refraction especially myopia In fatigue and "auto-intoxication"

CATARACT

The cataract is an opacity of the lens or its capsule It is not necessarily associated with significant decrease in vision

Etiology —The lens carries on an active metabolism The most important features in the development of cataract are diminished lens metabolism decrease in lens permeability and loss of substances active in oxidation such as cysteine glutathione and ascorbic acid Experimental cataracts have been produced by mechanical injuries physicochemical causes irradiation decreased permeability of the capsule interference with nutrient supplies anoxemia lack of sufficient proteins tryptophan or cysteine toxic effects of naphthalene lactose galactose thallium and dinitrophenols and parathyroidectomy

In man cataract is primarily associated with senility Factors that may participate in its development include heredity interference with the local nutrient supply of the lens general metabolic disturbances vitamin deficiency and an attack of rubella (p 417) in the first trimester of pregnancy

Developmental Cataract.—Developmental cataracts are lenticular opacities due to aberrations in the normal development of lens fibers and epithelium The polar cataract is a small central opacity which may or may not reduce vision to a significant extent Central cataracts occur near the sutures or in the fetal nucleus Punctate cataracts are generally of

fection are eliminated or drained Metabolic errors such as *uncontrolled* diabetes mellitus are remedied and hypertension is reduced if possible

In general it is better to avoid operation if vision is good in one eye The strong correction required after cataract extraction results in a 30 per cent enlargement of the image Glasses are poorly tolerated due to inability to fuse the larger image of the operated eye with that of the unoperated eye The surgical procedures performed for cataract are described elsewhere (p 1558) *Discissions* (p 1558) are advised in *congenital* and traumatic cataracts in patients below the age of thirty years *Intracapsular* or *extracapsular cataract extractions* are indicated in senile degenerative varieties An *iridectomy* (p 1588) may be wise as a preliminary to extraction in hypertensives and diabetics Secondary cataracts are managed by discission

LOCAL MANIFESTATIONS OF SYSTEMIC METABOLIC CONDITIONS

Metabolic disturbances of the organs of vision may be local manifestations of poisonings with drugs or chemicals or the effects of widespread metabolic abnormalities

OPHTHALMIC MANIFESTATIONS OF SYSTEMIC POISONINGS WITH DRUGS AND CHEMICALS

Acute Alcoholism (p 3848)
May be associated with incoordination of pupil impaired reaction to light or temporary amaurosis

Chronic Alcoholism (p 3851)
May cause toxic amblyopia or central scotoma May be avitaminosis (p 3266)

Methyl Alcohol (p 756)
May result in complete blindness with pupils dilated optic neuritis retinal hemorrhages and optic atrophy

Aniline (p 756)
May produce dark red fundus constriction of retinal vessels hemorrhage central scotoma and peripheral contraction of visual fields

Aminopyrine (p 3833)
Urticaria of lids is observed as idiosyncrasy

Arsenic (p 116)
Toxic amblyopia occurs especially with tryparsamide (p 120) *May result in optic atrophy and concentric contraction of peripheral* fields of vision

Aspidium (p 1895)
Bilateral amaurosis and optic atrophy as idiosyncrasy

Atropine (p 3875)
Paralysis of accommodation and mydriasis may precipitate acute glaucoma (p 1578)

Carbon Monoxide (p 747)
Overdose results in mydriasis ptosis ophthalmoplegia and progressive loss of central vision

diabetes and cholera There may be for instance *endocrine cataracts* in parathyroid tetany and myxedema cataracts with myotonic dystrophy myotonia congenita and Mongolian idiocy *dermatogenous cataracts* in neurodermatitis scleroderma and poikiloderma atrophicans *toxic cataracts* from dinitrophenol paradichlorobenzene (used as an insecticide or moth repellant) and ergot and *cataracta cachectica* which occurs in acute toxic illnesses

Complicated Cataract—The complicated cataract follows various intra ocular diseases It is presumably due to derangement of the metabolism of the lens by the diffusion into it of toxins from intra ocular fluids The main and earliest changes are seen in the region of the posterior pole of the lens This type of cataract follows usually severe ocular disease including iridocyclitis retinal detachment absolute glaucoma and intra ocular tumor

Secondary Cataract—Secondary cataract occurs in the remnants of the lens left behind after extracapsular operation or destruction by traumatism

Clinical Manifestations—The most important symptom of cataract is *diminished visual acuity* The degree of involvement depends upon the situation and extent of the cataract being greatest when it is central and diffuse Vision is best in dim light when pupils are dilated

At the onset of cataract the patient may complain of *black spots* which occupy a fixed position in the visual field *Polyopia* occurring in one eye is another frequent early symptom in which the images of objects are distorted or reduplicated due to irregular refraction within the lens This change also causes halos and stars around lights Myopia often develops in the early stages of cataract due to increased refractive power of the lens This second sight may cause the presbyopic patient to discard reading glasses

The objective signs of cataract consist of opacities in the lens Examination by *oblique illumination* (p 3622) shows a greyish or whitish opacity on a black background With the ophthalmoscope using a zero lens *at a distance a black opacity is seen on a red field If the cataract* is more or less complete the entire pupil appears greyish and the fundus reflex is absent

The complications of cataract are few A secondary rise in intra ocular tension may be due to swelling of the lens If the cataract is not removed when it is ripe and is allowed to become hypermature (*morgagnian cataract*) the hard nucleus of the lens floats about in the liquefied cortex Operation at this stage is difficult and rarely successful Unoperated the hypermature cataract may become dislocated and the eye may degenerate

Treatment—Attempts to stay the progress of lenticular opacities are difficult to evaluate since the course of cataract is variable and many never go on to maturity The local use of *dionin* (p 1548) has survived several centuries probably for want of something better A *high vitamin diet* with accessory feedings of vitamin B complex (p 682) may be recommended *Desensitization with lens protein has been tried but* the changes in the lens seem irreversible Very often a careful *refraction* and prescription for glasses will produce sufficient vision to postpone operation

Surgery affords the only positive means of curing cataracts Operative interference is preceded by thorough systemic investigation Foci of in

fection are eliminated or drained Metabolic errors such as uncontrolled diabetes mellitus are remedied and hypertension is reduced if possible

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Chronic Alcoholism (p 3851)
: May cause toxic amblyopia or central scotoma May be avitaminosis (p 3266)

Methyl Alcohol (p 756)
: May result in complete blindness with pupils dilated optic neuritis retinal hemorrhages and optic atrophy

Aniline (p 756)
: May produce dark red fundus constriction of retinal vessels hemorrhage central scotoma and peripheral contraction of visual fields

Aminopyrine (p 3833)
: Urticaria of lids is observed as idiosyncrasy

Arsenic (p 118)
: Toxic amblyopia occurs especially with tryparsamide (p 120) May result in optic atrophy and concentric contraction of peripheral fields of vision

Aspidium (p 1895)
: Bilateral amaurosis and optic atrophy as idiosyncrasy

Atropine (p 3875)
: Paralysis of accommodation and mydriasis may precipitate acute glaucoma (p 1578)

Carbon Monoxide (p 747)
: Overdose *results in mydriasis ptosis ophthalmoplegia and progressive loss of central vision*

Carbon Disulfide (p 747)
May cause photophobia nystagmus punctate keratitis impaired accommodation, micropsia macropsia and monocular diplopia

Bromide (p 3838)
Conjunctivitis

Caffeine (p 3866)
Amblyopia

Cannabis Indica
May result in impaired accommodation mydriasis visual hallucinations and amblyopia

Chloral (p 3837)
Miosis and later mydriasis from overdose

Chloroform (p 3925)
Mydriasis in profound poisoning

Chrysarobin (p 3118)
May cause bilateral conjunctivitis and keratitis

Cocainism (p 3916)
Visual hallucinations micropsia diplopia and amblyopia may follow

Digitalis (p 860)
Perversion of color sense

Dinitrophenol (p 754)
Cataract formation may result from obesity cures

Ergotism (p 240)
Spasm of retinal vessels pallor of optic disk neuroretinitis and cataract formation may result from overdosage or idiosyncrasy

Ether (p 3925)
Mydriasis from deep poisoning

Lead (p 762)
May cause extra ocular paralyses papilledema optic neuritis and hypertensive neuroretinitis

Mercury (p 765)
Retinal degeneration and hemorrhages may be produced

Methylene Blue
Greenish blue fundus

Morphine (p 3853)
Miosis toxic amblyopia and concentric peripheral contraction of visual fields are frequent sequels

Nitrobenzol (p 757)
See *Aniline*

Nitrous Oxide (p 4003)
Dilatation of retinal arteries

Optochin (p 204)
May cause amblyopia contraction of peripheral visual fields and central scotomas

Paradichlorobenzene (p 1592)
Cataract formation

Paraphenylendiamine (p 1592)
Diplopia lacrimation and chemosis may follow

Picric Acid
Yellow vision with loss of blue and violet

Phosphorus (p 729)
Retinal hemorrhages

Physostigmine (p 3874)
Miosis

Potassium Cyanide (p 748)
Fixed dilated pupils with exophthalmos

Quinine (p 519)
Sudden blindness amblyopia contraction of visual fields attentuation of retinal vessels and pallor of disk may result from idiosyncrasy or overdose

Resorcinol (p 3357)
Conjunctivitis

Santonin (p 1890)
Yellow vision

Silver (p 758)
Argyrosis of cornea in chronic poisoning

Salicylates (p 3340)
See *Quinine*

Sulfonamide (p 88)
May cause optic neuritis and transient myopia

Sulfonal (p 3841)
Ptosis extra ocular pareses and diplopia may result

Thallium (p 760)
Optic neuritis optic atrophy loss of brows and lids bilateral central scotoma and cataract formation may be produced

Tobacco (p 3884)
May cause amblyopia and angiospasms

Trichlorethylene (p 760)
Retrobulbar neuritis

Treatment—The appearance of ophthalmic defects possibly due to drugs and chemicals demands immediate discontinuance of the offending substance Antidotal therapy holds little promise sodium thiosulfate has

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May result in iritis conjunctivitis scleritis or tenonitis

Hemochromatosis (p 1976)
Slate blue pigment near disk

Hypertensive Toxemia of Pregnancy (p 2638)
May cause attenuation of retinal arteries angiospasms papilledema retinal hemorrhages and exudates or retinal detachment

Hyperthyroidism (p 1197)
May be associated with exophthalmos conjunctival edema exposure keratitis corneal ulceration paresis of extra ocular muscles infrequent blinking lid lag diplopia tearing pulsation of retinal arteries weakness on convergence and widened palpebral fissure

Hypoparathyroidism (p 1232)
Blepharospasm and cataract have been observed

Hypothyroidism (p 1191)
May produce puffy and edematous lids xerosis loss of outer third of eyelid or cataract

Jaundice (p 1951)
Sclera icteric

Laurence Moon Biedl Syndrome (p 1166)
Retinitis pigmentosa

Menstruation (p 2483)
Asthenopia edema and rings under eyes

Nephritis (p 2393)
Chemosis retinopathies and amaurosis

Niacin Deficiency (p 616)
Optic neuritis

Niemann Pick Disease (p 1134)
As Tay Sachs disease but yellow color to optic nerve

Normal Pregnancy (p 2617)
Pigmentations of eyelids

Pituitary Neoplasm (p 1153)
May result in bitemporal hemianopsia papilledema and optic atrophy

Schuller Hand Christian Disease (p 1135)
Usually associated with exophthalmos optic atrophy ulcerative keratitis and extra ocular muscle palsies

Tay Sachs Disease (p 1584)
Cherry red spot with white halo at macula and optic atrophy are characteristic

Thiamine Deficiency (p 616)
Conjunctivitis and oculomotor pareses may occur

been useless in our hands for the relief of *arsenical poisonings atropine mydriasis* cannot be alleviated by local instillation of pilocarpine and other miotics Injections of thiamine chloride using intravenous doses of 100 to 200 mg may have some efficacy in relieving the manifestations of *alcoholism* (p 3848) Angiospasmoses such as occur with *ergotism* and *nicotinism* may be relaxed by intravenous papaverine (p 3854) or aminophylline (p 3866)

OPHTHALMIC MANIFESTATIONS OF METABOLIC DISORDERS

Adrenal Cortical Deficiency (p 1271)
Enophthalmos

Allergy (p 1648)
May be associated with angioneurotic edema of eyes conjunctivitis vernal catarrh iritis optic neuritis or paresis of extra-ocular muscles

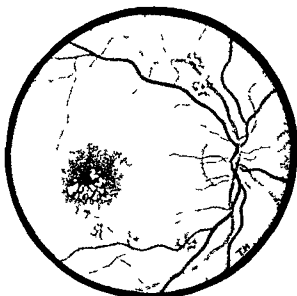


Fig 315 —Diabetic retinopathy *

Ariboflavinosis (p 616)
May cause rosacea keratitis with vascularization of cornea

Cevitamic Acid Deficiency (p 616)
Hemorrhages particularly retrobulbar may follow

Diabetes Mellitus (p 1246)
May produce errors in refraction and accommodation extra ocular palsies iritis cataract retinopathy hemorrhages retinitis proliferans optic and retrobulbar neuritis and lipemia retinalis with haziness and milky appearance of vessels

Gaucher s Disease (p 1133)
Pinguecula in nasal half of conjunctivae

* Gifford, Textbook of Ophthalmology

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Slate blue pigment near disk

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Vitamin A Deficiency (p 616)
May result in xerophthalmia keratomalacia light brown pigmentation of conjunctiva meibomitis blepharitis styes and night blindness

Xanthomatosis (p 1136)
Xanthelasma of lids

Treatment —A certain few of the ophthalmic manifestations of metabolic disorders may respond to specific measures of therapy The *water soluble vitamins* (thiamine chloride riboflavin niacin and cevitamic acid) are given intravenously in large doses for conditions that are possibly attributable to deficiency states *In vitamin A deficiencies* the lacking nutriment is given orally in massive dosage (p 690) *Allergic phenomena* respond symptomatically to injections or local instillations of epinephrine The early use of parathyroid hormone (p 1223) may prevent cataract formation in *parathyroid tetany* (p 1233), the ocular lesions of *hypothyroidism* regress with adequate doses of thyroid extract Emptying of the uterus may be followed by considerable reversal of the changes resulting from *hypertensive toxemia* (p 2638) Correction of the metabolic disorder in *diabetes mellitus* (p 1246) may prevent further damage but has little effect on conditions that have been established The *exophthalmos of hyperthyroidism* (p 1197) may recede after the use of iodides or deracil and following an adequate subtotal thyroidectomy (p 1214) In some instances it may progress despite best intended therapeutic efforts

CHAPTER 80

THE EYE: INFECTIONS INFLAMMATIONS ALLERGY

IDEAL management of ocular infection requires identification of the causative organism and localization of the anatomic site of the pathologic process Fully to realize the miracles of anti infective therapy the pathogen must be recognized so that its sensitivity or resistance to the various agencies can be determined Additionally rational therapy is dependent upon knowledge of the nature of the involved tissue Superficial infections of lids or conjunctiva are managed quite differently from similar invasions involving uveal tract or retina where the prognosis is infinitely graver The complete practitioner sets himself the problem of making a *simultaneous etiologic and anatomic diagnosis* Given the identity of the invading organism such as the gonococcus he sets out to discover whether he is dealing with a conjunctivitis susceptible to local treatment or an iritis which must be attacked by systemic methods Contrariwise given an anatomic diagnosis such as conjunctivitis he withholds specific therapy until he can determine whether he is dealing with an organism that is sensitive to sulfonamide penicillin or tyrothricin

In keeping with this view of the optimum method of dealing with ocular infection the current chapter has an initial subdivision devoted to the nature of the diverse invading organisms and specific bactericides The second portion of the chapter treats of the inflammatory processes as they are manifested at the various anatomic levels and serves to integrate specific and nonspecific methods of therapy

OCULAR MANIFESTATIONS OF SYSTEMIC INFECTION

OCULAR MANIFESTATIONS OF COCCAL INFECTION

Staphylococcus (p 151)

May cause *infection of superficial structures such as lids tear ducts*

and sacs and conjunctivae Particularly productive of blepharitis hordeolum and chalazion

Streptococcus (p 157)

May cause erysipelas involving lids cornea optic nerve orbit and meninges Scarlet fever may be complicated by conjunctivitis corneal ulcer and orbital cellulitis Episcleritis iritis and scleritis are encountered in rheumatic fever (p 186)

Pneumococcus (p 199)

May produce contact conjunctivitis or metastatic iritis dendritic keratitis and ophthalmia

Gonococcus (p 217)

Productive of ophthalmia in the newborn Metastatic iritis may be associated with bacteremia or focal infection particularly in the prostate

Meningococcus (p 208)

May produce isolated conjunctivitis or metastatic endophthalmitis ocular paralyses optic neuritis papilledema and amaurosis during bacteremia

Catarrhalis

May be associated with local infections of the lids and conjunctiva

Treatment —The specific treatment of coccal infection of the eye is an embarrassment of riches since the organisms are susceptible to sulfonamide and penicillin whether given locally or systemically Additionally tyrothricin is efficacious in gram positive invasions particularly with the pneumococcus

Superficial staphylococcal infections are best treated with 5 per cent sulfathiazole ointment or 30 per cent sodium sulfacetimide Resistant strains may require the substitution or addition of penicillin by instillation or intramuscular injection and when these agencies fail tyrothricin may be *topically administered*

Streptococcal infections involving the deeper structures or resulting from systemic invasion necessitate oral dosages of sulfonamide if the patient is ambulatory supplemented by parenteral penicillin *if there are* facilities for injection therapy Excellent results may be anticipated in erysipelas and scarlet fever but the rheumatic manifestations by their very nature are resistant

For *pneumococcic conjunctivitis* of contact origin ophthalmologists *place the greatest reliance on topical administration of* tyrothricin though local applications of sulfonamide and penicillin may be equally successful Deeper involvements must be managed by oral doses of sulfonamide or parenteral administration of penicillin

Infections with *gonococcus* or *meningococcus* seem best treated by intramuscular injections of penicillin Satisfactory results also may be obtained in all but infections with resistant strains by topical and oral administration of sulfonamide

OCULAR MANIFESTATIONS OF BACILLARY INFECTION

The significance of tuberculous infection of the eye overshadows the remaining bacillary disturbances of the organs of vision None of the ophthalmic structures displays significant immunity to the acid fast invader and experienced ophthalmologists suspect tuberculosis in any chronic inflammatory process particularly if it occurs in a patient who is known to have or have had a Koch bacillus infection elsewhere in the body

B anthracis (p 299)
Malignant pustule may occur around the eyelids

C diphtheriae (p 302)
May cause conjunctivitis optic neuritis post diphtheritic extra ocular palsies and paralysis of accommodation

S dysenteriae (p 243)
Conjunctivitis and iridocyclitis are rare complications

H influenzae (p 396)
May produce conjunctivitis purulent dacryocystitis herpetic keratitis iritis uveitis retinal hemorrhages ocular palsies and optic neuritis

M leprae (p 273)
Leprosy may be associated with anesthetic patches on the lids loss of lashes and eyebrows nodular deformities of the lids conjunctivitis superficial keratitis nodular iritis and optic atrophy

M mallei (p 327)
Glanders may be represented by a primary implant with a pre auricular lymphadenopathy or conjunctivitis

B melitensis (p 314)
Brucellosis may cause iritis iridocyclitis optic neuritis papilledema optic atrophy extra ocular palsies conjunctivitis and hemorrhagic phenomena

H pertussis (p 278)
Cough may be followed by sub conjunctival and orbital hemorrhages

P pestis (p 321)
Plague may be associated with exudative retinitis and retinal detachment

M tuberculosis (p 252)
The tubercle bacillus may produce choroid tubercles ulcero necrotic conjunctivitis sclerosing keratitis interstitial keratitis uveitis retinitis retinal periphlebitis (Eales disease) phlyctenular keratoconjunctivitis (p 1628) uveo parotid fever (Heerfordt s disease) (p 1635) chorioretinitis juxtapapillaris (Jensen s disease) (p 1635) and Boeck s sarcoid with iritis conjunctivitis and uveitis (p 3271)

P tularensis (p 323)

The primary lesion of tularemia may occur in the conjunctiva as an ulcero necrotic nodule with regional lymphadenopathy

E typhosa (p 225)

Typhoid fever may be characterized by corneal ulceration iritis metastatic choroiditis optic neuritis ocular palsies and retinal hemorrhages

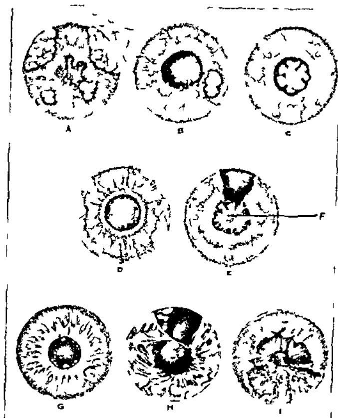


Fig 316—A Peracute conglomerate tuberculous iridocyclitis with perforation of the sclera B Solitary tuberculoma C Subacute nodular or follicular tuberculous iritis D Tuberculous iritis of the ciliary region glassy walls of lymphomatous tuberculous nodules E Tuberculous iritis involving the pupillary area F Occlusion of the pupil G Knobs on the pupillary border in quiet cyclitis H Quiet tuberculous iridocyclitis with mutton fat precipitates and atrophic changes I Anterior synechia after iris bombee and cured tuberculosis

V comma (p 249)

Sunken eyeballs and dullness of the cornea result from the dehydration of cholera

Treatment—Anti infective treatment of bacillary infection of the ocular apparatus is disappointing The sulfonamides and penicillin may be administered locally and parenterally in certain superficial inflammatory processes such as the malignant pustule of anthrax Lesions produced by P tularensis H influenzae H pertussis M tuberculosis and the typhoid

Zeeman in Berens The Eye and It Diseases

dysentery group merit therapy with streptomycin (p 106) by topical or parenteral administration Chemotherapy may be given in conjunction with serum therapy in anthrax and diphtheria but the brilliant successes seen in coccal infection cannot be duplicated

OCULAR MANIFESTATIONS OF SPIROCHETAL INFECTION

The lesions of syphilis dominate ocular spirochetal infections Particularly in a known syphilitic the presence of chronic inflammation suggests a local ocular manifestation of systemic invasion Nevertheless the practitioner must remember that the syphilitic is as prone to develop non syphilitic ocular disturbances such as cataract or glaucoma as his Wassermann negative fellows

L icterohemorrhagiae (p 360)
Infectious jaundice may be associated with conjunctivitis iritis irido cyclitis retinal hemorrhages optic neuritis and icterus of the sclera

Sp minus (p 363)
The primary inoculum of rat bite fever may occur on the lids Later the patient may develop conjunctivitis and uveitis

B recurrentis (p 357)
Uveitis iridocyclitis and retinal hemorrhages may be noted in relapsing fever

T pallidum (p 331)
Syphilis may become manifest through a chancre of the lids orbital periostitis extra ocular paralyses internal ocular paralyses (Argyll Robertson pupil) optic neuritis papilledema primary optic atrophy scleritis interstitial keratitis (in neonatal infection) iritis and choroiditis (p 1632)

T pertenue (p 351)
Yaws may be accompanied by granular conjunctivitis and secondary contractions resulting in ectropion and exposure keratitis

Treatment—The acute inflammations resulting from spirochetal invasion of the visual apparatus may be expected to yield with miraculous efficacy to the arsenicals (p 116) and penicillin (p 106) The chronic processes do not present a favorable prognosis since many represent irreversible lesions such as optic neuritis Additional therapeutic measures worthy of trial under these circumstances include hyperthermia malarial therapy and injections of tryparsamide

OCULAR MANIFESTATIONS OF RICKETTSIAL AND VIRUS DISEASES

Rickettsial disease is of minor importance in ophthalmic infection but ocular tissues appear to be highly susceptible to virus invasions whether by local implant or systemic dissemination The predominantly local virus infections of the eye are described in detail with the material on conjunctivitis (Beal s disease epidemic keratoconjunctivitis inclusion conjunctivitis and trachoma p 1695) The local manifestations of systemic virus disease are listed below

Anterior Poliomyelitis (p 457)
Bulbar type results in paralyses of extra ocular muscles

Chickenpox (p 420)
Vesicles may appear on the lids conjunctiva and cornea with progression to secondary iritis

The Common Cold (p 391)
Usually associated with suffusion of the conjunctiva and nasolacrimal edema

Epidemic Encephalitis (p 441)
May result in diplopia ptosis paralysis of extra ocular muscles nystagmus Argyll Robertson pupil disturbances of associated movement loss of convergence and paralysis of divergence

Foot and Mouth Disease (p 435)
Often results in conjunctivitis and corneal ulceration

Herpes Febrilis (p 433)
Painless groups of vesicles may appear on lid Healing occurs without *scar formation*

Herpes Zoster (p 435)
Herpes ophthalmicus is a serious infection accompanied by severe pain and vesiculation Lesions of the upper lid accompany involvement of the first division of the trigeminal nerve those of the lower lid indicate involvement of the second division with possible implication of the eyeball Ulceration of vesicles may lead to insensitivity of the cornea iritis, uveitis secondary glaucoma and loss of vision

Lymphopathia Venereum (p 471)
May be associated with follicular conjunctivitis pre-auricular lymphadenopathy uveitis retinal hemorrhages and episcleritis

Measles (p 409)
Early suffusion of lids with conjunctivitis Later keratitis with corneal ulceration may result in permanent impairment of vision Infrequent complications include weaknesses of accommodation optic neuritis and extra ocular palsies

Molluscum Contagiosum (p 422)
Produces umbilicated tumors of lid margins with conjunctivitis

Rubella (p *417*)
In first trimester of pregnancy may produce cataract of the newborn and retrolental fibroplasia

Smallpox (p 424)
The pustules of cornea may result in ulceration residual opacity impairment of vision

Typhus (p 369)
May be accompanied by exudative and hemorrhagic retinitis

Vaccinia (p 428)
Auto contamination of inoculum may produce lesions of lids con

junctiva and cornea Under latter circumstance ulcerative keratitis and impairment of vision may be produced

Treatment—The treatment of the local virus diseases of the eye is discussed with the material on conjunctivitis (p 1616) Encouraging results have been obtained with sulfanilamide in the management of inclusion conjunctivitis and trachoma The ocular manifestations of systemic virus disease present a less rosy prospect but rickettsial infections yield to para aminobenzoic acid

OCULAR MANIFESTATIONS OF MISCELLANEOUS INFECTIONS

The practitioner rarely encounters ophthalmic manifestations of invasions with fungi protozoa and helminths but an important problem is posed by disturbances of the visual apparatus due to focal infection and to spread by continuity particularly from the accessory nasal sinuses The investigation of orbital cellulitis and inflammations of the vascular tunic and neural elements is incomplete unless there has been a meticulous survey of teeth upper respiratory passages and genital tract for the possible presence of a more fundamental lesion

Actinomycosis (p 489)
May cause orbital involvement secondary to lumpy jaw

Acute Disseminated Lupus Erythematosus (p 3599)
Produces eruption on lids and hemorrhages and exudates of retina

Aspergillosis (p 498)
Corneal ulceration is occasionally encountered

Blastomycosis (p 493)
May involve skin of eyelids and conjunctiva Rare iritis

Dermatomyositis (p 3373)
May be associated with pareses of external ocular muscles nystagmus exophthalmos iritis retinitis flame shaped retinal hemorrhages

Erythema Nodosum (p 3377)
Nodular swellings may involve subconjunctival regions

Favus (p 3304)
Rare conjunctivitis but frequent involvement of lids

Filariasis (p 3326)
Parasites in intra ocular tissues or conjunctival sac (Blinding Filaria)

Focal Infection (p 42)
Of particular importance in keratitis iritis iridocyclitis choroiditis uveitis optic neuritis retrobulbar neuritis and scleritis

Infection by Continuity (p 2130)
Inflammation of accessory nasal sinuses of greatest significance in orbital cellulitis optic neuritis and uveitis

Leishmaniasis (p 534)
May cause interstitial keratitis corneal abscess and perforation

Leptothricosis (p 1623)
Granulations on conjunctiva and enlargement of pre auricular gland (Syndrome of Parinaud) with demonstrable leptothrix

Malaria (p 507)
Dendritic corneal ulcer with retinal hemorrhages Quinine amaurosis

Myiasis
Larvae in conjunctivae cause inflammation and occasional corneal ulceration Larvae in vitreous may cause blindness

Pediculosis (p 3131)
Nits involve lashes

Schistosomiasis (p 537)
Conjunctival tumors may contain ova

Sporotrichosis (p 495)
Rare conjunctivitis or keratitis

Teniasis (p 1899)
Eggs may be deposited in any structure of eye causing severe local inflammation

Thrush (p 1697)
Rare conjunctivitis

Trichinosis (p 539)
Chemosis subconjunctival hemorrhage and limitation of movements of eyeball

Trichophytosis (p 3293)
Rare conjunctivitis or keratitis

Trypanosomiasis (p 531)
Keratitis of interstitial type with vascularization Uveitis also occurs

Treatment —Local treatment may be of value in many of the miscellaneous inflammatory processes involving the superficial ocular structures Included in this group are the fungous disturbances filariasis myiasis pediculosis teniasis and trichophytosis Most dramatic therapeutic successes are often obtained by elimination of foci of infection and the control of neighboring disturbances particularly the spheno ethmoiditis that is associated with optic neuritis *In the investigation of these latter problems* the practitioner should resort to round table consultation with ophthalmologist and rhinologist

PREDOMINANTLY LOCAL INFECTIONS, INFLAMMATIONS AND ALLERGIES OF THE VISUAL APPARATUS

Infections inflammations and allergies may result in specific elective involvement of the several tissues that make up the ocular structures Prognosis and therapy are greatly influenced by the site of the lesion as illustrated in the remaining material of the present chapter

DERMATITIS OF THE LIDS

See *Analogues of the Dermatoses* (p 1564)

ABSCESS OF THE LIDS

Abscess of the lids usually follows injury or infection of the eyebrows particularly after plucking The free borders of the lids and the cilia are not usually involved even when the abscess is extensive

Continuous *hot wet dressings* and local applications of 5 per cent sul fathiazole ointment are of great value in the early course of the abscess When fluctuation develops a *horizontal incision* is made parallel to the lines of the skin The venous return of the upper lid into intracranial chan nels suggests the wisdom of instituting prophylactic chemotherapy with *sulfonamide* (p 88) and/or penicillin (p 106)

ULCERS OF THE LIDS

Ulceration of the skin of the lids has ominous implications The lesion may be *vaccinal* (p 428) following immunization but on rare occasions it represents the primary infection of *syphilis* (p 331) *tularemia* (p 323) or *rat bite fever* (p 351) Some lesions are *epitheliomatous* (p 3220) and others are late manifestations of *leprosy* (p 273) or *tuberculosis* (p 252)

The differential diagnosis of ulcerations of the lids must be intensively pursued The recognition of rat bite fever is apparent from the history Tularemia is suggested in patients bitten by deer flies or ticks and in those who have handled the hides of infected rabbits and other rodents Dark field examination (p 45) is required for the identification of the *T pal lidum* of syphilis and if this test is not definitely positive a biopsy is in dicated to reveal the nature of the inflammatory or neoplastic process

BLEPHARITIS MARGINALIS

Blepharitis marginalis or ciliaris is an inflammation of the margin of the lid It is a very common and recurrent condition characterized by local redness thickening granulation scaling and crusting Itching local sore ness and asthenopia are important symptoms but most frequently the pa tient seeks medical care for cosmetic reasons

The disturbance starts as a simple hyperemia of the lid margin and often is seen in persons of fair complexion In extreme examples the edges of the lids are swollen and yellowish crusts glue the lashes together to cover bleeding ulcers Eventually the lashes fall out or become distorted due to destruction of hair follicles Lid margins become greatly thickened and *trichiasis* and *ectropion* occur (p 1569) *Chronic conjunctivitis* (p 1616) and *recurrent styes* (p 1610) are frequent complications

Etiology—In blepharitis the usual causative organism is *Staphylococcus aureus* or toxic *Staphylococcus albus* (p 151) *Predisposing factors* are said to include errors of refraction poor resistance to infection poor hygienic surroundings adverse occupational conditions irritation from cosmetics and defects in diet Most often in our experience the lesion is independent of these circumstances and seems to occur on some presently unknown constitutional basis

Treatment—Local treatment includes the application of 1 or 2 per cent silver nitrate to the lid margins the *manual expression* of lid glands and removal of scales crusts and diseased cilia Ointments of 1 3000 *bichloride of mercury* *penicillin* or 5 per cent *sulfathiazole* are applied locally If the condition persists or recurs a culture of the lid

margin is taken and if a pathogenic staphylococcus is recovered injections of *staphylococcus toxoid* (p 78) or *an autogenous vaccine* are given The senior author is convinced that there is a close association between blepharitis and seborrhea sicca (dandruff) It is his experience that the lids will not clear unless scalp treatments are undertaken at the same time (p 3137)

PHTHIRIASIS PALPEBRARUM

Phthiriasis palpebrarum the presence of the *crab louse* (pediculosis pubis) on the eyelashes usually occurs in children and responds readily to inunctions of 3 per cent *ammoniate mercury ointment* (p 3137)

HORDEOLUM (STYE)

Hordeolum is an *acute suppurative inflammation* of one of the glands of Zeis on the lid margin As a result the affected lid and contiguous conjunctival membrane are swollen and the pre auricular gland enlarges *Gentle palpation of the lid margin with a cotton applicator* reveals an espe

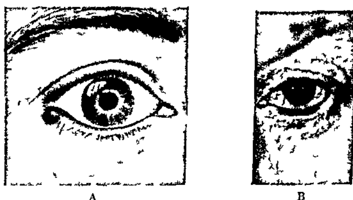


Fig 317—*A* Hordeolum *B* Chronic blepharitis

cially sensitive spot corresponding to the inflamed gland Within a day or two increasing swelling redness and yellowish discoloration are noted in the area

Etiology—Styes or external hordeola are due to *staphylococcic infection* (p 151) While they occur at all ages they are most common in children or young adults and often appear in crops They are frequently an expression of a lowered state of resistance due to errors in diet or hygiene and they may be associated with an *uncorrected refractive error* In the middle aged recurring styes are seen in uncontrolled *diabetes* (p 1246)

Treatment—*In the incipient stage symptoms may subside after removal of a lash* in the region of the involved gland and applications of penicillin or *5 per cent sulfathiazole ointment* On failure of abortive treatment *hot compresses* are used to bring the stye to a head At this time the conservative wait for the abscess to evacute but the less patient release pus by a horizontal incision using a sharp scalpel It is the experience of the senior author that the policy of watchful waiting' is

followed by fewer recurrences since abscess fluid is more likely to be sterile at the time of spontaneous release

If styes tend to recur injections of *staphylococcus toxoid* (p 78) or *autogenous vaccine* are used if cultures reveal the presence of a toxic staphylococcus The patient is referred to the specialist for the correction of refractive errors but despite best efforts lesions may continue to recur

CHALAZION

Chalazion is a chronic inflammatory enlargement of a meibomian gland A hard swelling of the lid may develop without gross inflammatory symptoms until it reaches the size of a small or large pea The tumor is circumscribed and adherent to the tarsus but not to the skin Eversion of the lid reveals reddening and thickening of overlying conjunctiva which later assumes a grayish yellow appearance In this phase the chalazion is unsightly and productive of conjunctival irritation Eventually there is cyst formation or granulation of the mucosal surface

With secondary infection an internal hordeolum or suppurating chalazion appears The lids become red and swollen the conjunctiva is chem

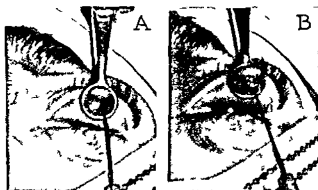


Fig 318—Technic of operation for chalazion A Incision B Curettage

otic pain is so severe that it may be impossible to evert the lid in order to observe the conjunctiva

Treatment—The treatment of the *non infected chalazion* is accomplished by surgical excision (p 1556) If the *secondarily* infected chalazion is seen within the first few days of its onset suppuration may be prevented by expression of discharge through the opening of a meibomian gland Hot compresses are applied and penicillin or 5 per cent sulfathiazole ointment is rubbed into the area In the presence of *associated constitutional symptoms* and with a *hyperacute* or *spreading local lesion* systemic administration of sulfonamide or penicillin (p 106) is advised

When the infected chalazion has localized pus is evacuated through a vertical incision of the conjunctiva or one that is horizontally placed through the skin The lesion promptly subsides with adequate drainage but residual granulation tissue may require attention at a later period Cultures usually reveal a pathogenic staphylococcus

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Eyelashes and Eyelids

Abnormalities involving the ocular adnexa are usually the concern of the practitioner to whom the patient appeals particularly in communities where there are no special st oph thalmologists

	DIAGNOSTIC FEATURES
Congenital Anomalies	See p 1560
Analogues of the Dermatoses	See p 1564
Physical Mechanical and Chemical Injuries	See p 1569
Cysts and Neoplasms	See p 1566
Inflammations and Infections	Particularly blepharitis hordeolum (tye) and chalazion
Allergies	Contact dermatitis from ocular medications or *nail polish*
Shaggy Eyebrows	Familial Acromegaly with prognathus and changes in sella turcica
Scant Eyebrows	Hyperpituitarism with giantism or acromegaly *Leprosy with anesthetic nodules* Albinism with lack of pigment Myxedema with low B M R. and therapeutic response to thyroid extract
Lagophthalmos (Inability to Close Eye)	Facial paralysis Exophtha mos
Ptosis (Drooping of Upper Lid)	See p 1649
Crusting of Lids	Hyper ecretion of glands of Moll Blepharitis marginalis Asthenopsia with refractive error Seborrhea (dandruff) Hordeolum (stye) Contact dermatitis Measles with character istic xanthem
Widening of Palpebral Fissure	With exophthalmos myopia orbital cellulitis and cavernous sinus thrombosis Make local examination *Refract and perform exophthal mometry* (p 1546)
Narrowing of Palpebral Fissure	Enophthalmos particularly in Horner s syn drome Dehydration ptosis and blepharo phimosis
Dark Circles Under Eyes	Familial With fatigue menstruation preg nancy senility hyperthyroidism and adrenal cortical deficiency
Blepharospasm	With burns foreign bodies allergies con junctivitis keratitis hypoparathyroidism, as thenopsia habit spasm chorea and albinism Make local inspection and obtain exudate for smears and cultures Get epithelial scrapings (p 1546)
Ecchymosis (Black Eye)	Following direct trauma fracture of skull per tussis epilepsy and hemorrhagic diatheses If no direct injury get skull x ray and hemo gram (p 3704)
Ulcers of Lids	Blepharitis marginalis epithelioma primary syphilis tularemia and glanders In doubt examine by darkfield microscopy and by direct smears and cultures (p

D

Edema of Eyelids	Insect bites and burns Inflammations and infections of lids conjunctiva sclera cornea lacrimal passages and lacrimal glands Contact dermatitis particularly with cosmetics Glaucoma with increased intracranial tension Orbital cellulitis and panophthalmitis Cavernous sinus thrombosis with bacteremia Nasal accessory sinusitis particularly of sphenoids and ethmoids with x ray changes Pertussis with paroxysmal cough Measles varicella and variola with characteristic rash Trichinosis with eosinophilia Angio neurotic edema of allergic origin Hypothyroidism with low B M R Anemia with characteristic hemogram (p 3704) Nephropathies with characteristic urinary and blood findings (p 2362) Backward failure Nephrosis Water intoxication Salt retention and dermatitis medicamentosa (p 3335)

With recurrence *preventive measures* are instituted The patient is referred to the ophthalmologist for refraction Errors in general hygiene are corrected Injections of staphylococcal toxoid or of autogenous vaccine are begun and 5 per cent sulfathiazole ointment or 1 per cent yellow oxide of mercury ointment is massaged into the lids each night

CHRONIC MEIBOMITIS

Chronic infections of the meibomian glands result in redness and swelling of the lid margin and the appearance of foamy secretion in the conjunctival sac

The lids are expressed frequently to prevent multiple *chalazia* and *blepharitis* The patient is advised to practice nightly inunctions of *5 per cent sulfathiazole* or 0.5 per cent *ammoniated mercury*

TARSITIS

Involvement of the tarsus may result from *syphilitic tuberculous* or *trachomatous infection* The condition becomes manifest as a thickening of the lids with tenseness and redness of the overlying skin With positive serologic tests for syphilis antiluetic therapy (p 340) is instituted Otherwise the patient is referred to the specialist for diagnosis if necessary by examination of smears and biopsy

Local treatment is palliative Hot compresses and antiseptic ophthalmic ointments are used twice daily

DACRYADENITIS

Acute inflammation of the tear glands is extremely rare When present it is manifested by swelling redness and tenderness of the upper temporal margin of the orbit The lesion is treated in the manner of a suppurating chalazion (p 1611) preferably by the consulting ophthalmologist

Mikulicz s Disease —Chronic bilateral enlargement of the lacrimal glands associated with similar processes in the parotid and other salivary glands occurs in Mikulicz s disease (p 1709) The latter is often associated with *leukemia* (p 1100) so that complete blood studies are required

CHRONIC DACRYOCYSTITIS

Chronic inflammation of the lacrimal sac usually follows obstruction of the nasal duct (p 1569) With simple occlusion, the only symptom is tearing With distention and nonpurulent inflammation a *mucocele* develops in the region of the sac and watery or mucoid fluid escapes from the punctum In chronic purulent dacryocystitis pressure upon the distended sac yields pus containing staphylococci streptococci pneumococci or the Friedlander bacillus Persistence of purulent dacryocystitis results in *chronic conjunctivitis* (p 1616) and the threat of serious *corneal inflammation*

Treatment —The patient with dacryocystitis is referred to the consultant ophthalmologist Non operative procedures are attempted by *dilatation and irrigation of the lacrimal passages* (p 1557) If the disturbance fails to subside operative interference is advisable *Dacryocystectomy* (p 1557) is the simpler procedure but it may be followed by persistent tearing The more extensive *dacryocystorhinostomy* (p 1557) in which the cavity of the lacrimal sac is joined directly with the nasal fossa, is a more formidable procedure but yields more satisfactory late results

ACUTE DACRYOCYSTITIS

Acute dacryocystitis almost always follows the chronic form of infection (above) On rare occasions it results from extension of infection from the nose or its accessory sinuses The lesion is more properly termed an *acute peridacryocystitis* since it is really a purulent inflammation of the connective tissues that surround the chronically inflamed tear sac

The condition is recognized by swelling redness and edema of the skin that overlies the lacrimal sac There may be associated chemosis pain tenderness and constitutional symptoms Suppuration is manifested by the appearance of a localized area of yellowish discoloration

Treatment —In the early phases of an acute dacryocystitis the lesion is treated with continuous wet dressings The appearance of constitutional symptoms is sufficient indication for systemic chemotherapy using *sulfonamide* (p 88) or *penicillin* (p 106) If the inflammation does not subside the abscess is drained by *incision* which may require induction of *general anesthesia* Delay in evacuation of pus may lead to the formation of a *lacrimal fistula*

ORBITAL PERIOSTITIS

Inflammation of the bone of the orbit and orbital periosteum may be acute or chronic localized or extensive Orbital periostitis usually results from injury tuberculosis syphilis or infection of the nasal accessory sinuses The margin of the orbit is most often involved *Symptoms* consist of localized pain tenderness and swelling A *subperiosteal abscess* may develop with later formation of a *fistula* If the periostitis occurs posteriorly it simulates an *orbital cellulitis* (p 1615) and is treated as such

Treatment —The patient with orbital periostitis requires hospitalization Serologic tests for syphilis are performed radiographs of the skull and nasal accessory sinuses are required and ophthalmologist and rhinologist are summoned in consultation

In the presence of positive evidences for syphilis antiluetic therapy is instituted (p 340) Nonsyphilitic infections are treated systemically with

sulfonamides and penicillin (p 106) Suppuration in the nose or accessory sinuses requires indicated operative procedures

ORBITAL CELLULITIS

Orbital cellulitis may be secondary to *erysipelas* (p 167) or *orbital periostitis* More often however it is observed in children with purulent sinusitis involving *frontals sphenoid* or *ethmoids* (p 2125)

Orbital cellulitis is manifested by inflammatory edema of the lids chemosis protrusion and/or displacement of the eyeball limited ocular motility and diminution of vision if there is involvement of the optic nerve

The course of orbital cellulitis is variable The condition may subside with absorption of exudate or it may progress to suppuration and result int *orbital abscess* The latter requires prompt *evacuation* to prevent seri

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Orbit

Disturbances of the orbit are always of sufficient significance to warrant specialist consultation

	DIAGNOSTIC FEATURES
Congenital Anomalies	See p 1560
Mechanical Disturbances	Exophthalmos and enophthalmos
Neoplasms	See p 1567
Infections	Orbital cellulitis periostitis osteomyelitis and abscess Get x rays of orbit and nasal accessory sinuses Consult rhinologist and ophthalmologist
Vascular Disturbances	Cavernous sinus thrombosis with bacteremia Occlusion of retinal arteries and veins visible by ophthalmoscopy Arteriovenous aneurysm of internal parotid artery and cavernous sinus with pulsating exophthalmos

ous sequels such as optic neuritis panophthalmitis purulent meningitis or thrombosis of the cavernous sinus (p 2130)

Treatment —The patient with orbital cellulitis requires immediate hospitalization and consultations are required with ophthalmologist and rhinologist *Chemotherapy* with sulfonamides and/or penicillin (p 106) is instituted but not to the exclusion of necessary *surgical procedures* such as drainage of the orbital abscess or operations designed for the relief of the more fundamental suppurative processes in the nose and its *accessory* sinuses

TENONITIS

Inflammation of Tenon s capsule occurs rarely and is of obscure etiology It may be associated with gouty and rheumatic diatheses Tenoniti may follow *operations for squint* (p 1530) due to contamination of the

operative field It is characterized by extreme chemosis and limitation of ocular motion without displacement of the eyeball

The management of a tenonitis is specialist province Injections of foreign protein and anti infective therapy may be tried With arthropathies salicylates may be useful

CONJUNCTIVITIS

Inflammations of the conjunctiva are extremely common and may be caused by a variety of organisms The severity ranges from mere annoyance to *total blindness*

Etiology—Infection of the conjunctiva may be a purely *local invasion* On rare occasions the inflammatory process is of *metastatic origin* in the course of systemic disease and the conjunctival lesion may represent a portal of entry resulting in generalized dissemination of the invading *organism*

The list of those organisms which may produce local conjunctivitis includes staphylococcus, streptococcus pneumococcus meningococcus gonococcus M tuberculosis M leprae H influenzae H conjunctivitidis (Koch Weeks) H duplex (Morax Axenfeld) C diphtheriae T pertenue (yaws) the viruses responsible for inclusion conjunctivitis trachoma Beal s follicular conjunctivitis and epidemic keratoconjunctivitis leptothrix and streptothrix

Metastatic conjunctivitis is most often encountered in the exanthematous diseases (roseola rubella and scarlet fever) gonorrhea epidemic cerebrospinal meningitis and bacillary dysentery Conjunctival contamination may result from auto inoculation in vaccinia variola and varicella Finally the conjunctival lesion may prove to be a portal of entry when there is a primary infection due to syphilis, tularemia or glanders

Pathology—The pathology of conjunctivitis is studied under the magnification of the slit lamp Catarrhal membranous purulent and follicular *inflammations are observed*

Catarrhal Conjunctivitis—Catarrhal conjunctivitis is seen in acute subacute and chronic phases Commoner etiologic causes are *pneumococcus Staphylococcus aureus H influenzae H duplex* (Morax Axenfeld) *H conjunctivitidis* (Koch Weeks) and *Streptococcus haemolyticus*

The *acute phases* are marked by hyperemia mucopurulent discharge redness and swelling of the mucous membranes and the appearance of vascularized papillae in the tarsal region Acute catarrhal conjunctivitis has a marked tendency toward recovery although occasionally it passes into a subacute or chronic phase *Subacute varieties* are frequently due to the Morax Axenfeld bacillus and *chronic lesions* are often associated with the presence *of precipitating factors such as local irritation disturbances* in general hygiene avitaminoses malnutrition and alcoholism Chronic conjunctivitis is often associated with a *chronic blepharitis* (p 1609) and results in the formation of fine infiltrations of the cornea

Membranous or Pseudomembranous Conjunctivitis—Membranous or pseudomembranous conjunctivitis is observed as a result of infections by *Streptococcus haemolyticus* or *C diphtheriae* In either instance the inflammatory process is severe and is often followed by necrosis of the cornea and subsequent scar formation The gravity of membranous conjunctivitis warrants *specialist consultation*

Purulent Conjunctivitis—Purulent conjunctivitis is most frequently of *gonorrheal origin* (p 217) The inflammatory process is hyperacute and may terminate in *blindness* due to corneal involvement Recovery is often associated with *residual opacities* and *cicatricial deformities* which seriously interfere with vision (p 1638)

Follicular Conjunctivitis—The follicular types of conjunctivitis most often are *viral* in origin In *inclusion conjunctivitis* (p 1623) and *trachoma* (p 1625) it is possible to demonstrate *inclusion forms* in the epithelial scrapings (p 1546) Bacterial stains fail to reveal the presence of the ordinary organisms In trachoma and epidemic keratoconjunctivitis the mucous membrane lesion is accompanied by corneal involvement healing is associated with opacification and diminution of vision

Clinical Manifestations—The generic manifestations of conjunctivitis include a scratchy or sandy feeling in the eyes itching smarting and photophobia Occasionally the onset is announced by sharp *pain* that suggests the presence of a foreign body In the more severe infections the lids feel hot and heavy and large amounts of exudate may cause blurring of vision



A



B

Fig 319—*A* Acute catarrhal conjunctivitis *B* Acute gonorrheal conjunctivitis

Most of the acute examples of conjunctivitis are associated with enlargement of the pre auricular lymph nodes

In *acute catarrhal varieties* the discharge is mucopurulent the mucous membrane is red and swollen and smears and cultures reveal the nature of the causative organism Profuse discharge characterizes the *purulent forms* of conjunctivitis By contrast in *follicular types* the discharge is scant but conjunctival hyperemia congestion and tearing are intense

The course complications and management of conjunctivitis are dependent for the most part upon the nature of the invading organism as described in succeeding paragraphs

Differential Diagnosis—The initial diagnostic problem in acute conjunctivitis is the differentiation of the mucous membrane infection from *disturbances of the uveal tract* (p 1618) and the syndrome of an *acute glaucoma* (p 1578)

Once the diagnosis of conjunctivitis has been established it is necessary to determine the nature of the *causative organism* (p 1619) and the

TABLE 110—DIFFERENTIAL DIAGNOSIS OF CONJUNCTIVITIS IRITIS AND GLAUCOMA

	Acute Conjunctivitis	Acute Iritis	Acute Glaucoma
History			
Onset	Usually gradual	Usually gradual	Sudden
Pain	Discomfort sandy feeling but no real pain	Moderate pain in eye radiating to forehead and temple	Very severe pain in eye radiating over entire sensory distribution of nerve V
Systemic complications	No systemic complications	No systemic complications	Vomiting and prostration
Vision	Good	Moderate impairment of vision	Marked impairment of vision
Pre auricular gland	Often enlarged palpable and tender	Not palpable	Not palpable
Secretion	Usually mucopurulent although may be watery Lids stick together after sleep Smear often shows organisms	None or watery	None or watery
Injection	Superficial or conjunctival injection	Ciliary or deep injection	Ciliary or deep injection
Conjunctiva	Palpebral conjunctiva reddened thickened and hyperplastic	*Usually normal*	Chemosis often present
Cornea	Clear	Clear but may show deposits on posterior surface	Steamy and insensitive
Iris	Clear	Lacking in luster	Muddy with green sheen
Anterior chamber	Normal	Normal depth but cloudy due to exudation	Shallow
Pupil	Normal	Small and irregular	Dilates oval and fixed
Tension	Normal	Usually normal or low	Increased
Ocular tenderness	None	Marked	Very marked
Fundus	Normal	Normal	Glaucomatous cupping

cytology of the secretion Identification of the particular culprit establishes prognostic implications and the therapeutic program

Complications —Conjunctivitis may be associated with local and systemic complications The latter are rarely observed since the conjunctiva acts as an effective barrier to prevent dissemination of infection Exceptions consist of instances of primary infections in ***syphilis*** (p 331) *tularemia* (p 323) and ***glanders*** (p 327)

The *local complications* of conjunctivitis may be immediate or delayed The immediate hazard is the occurrence of corneal involvement such as particularly occurs in infections with pneumococcus gonococcus and the viruses of keratoconjunctivitis and trachoma

Late aftermaths are the effects of scarring corneal opacities are seen in keratoconjunctivitis trachoma and gonorrheal ophthalmia Conjunctival deformities are most frequent in trachoma and yaws

Treatment —Conjunctivitis may be treated by preventive and active measures the latter include local topical and systemic methods of therapy

Prophylaxis—Active measures to prevent conjunctivitis are routine procedures in the newborn and in the care of physicians and other medical attendants whose eyes have been contaminated by infectious discharge Before the introduction of newer anti infective agents it was common procedure to irrigate the eyes with normal saline and then instill 1 to 2 drops of 0.5 to 1 per cent silver nitrate After a wait of at least a minute the eyes were again irrigated with saline solution Despite best efforts therapy was often followed by a chemical conjunctivitis of annoying severity so that 25 per cent mild silver proteinate and 10 per cent strong proteinate were often substituted

TABLE 111 —LABORATORY AIDS IN THE DIAGNOSIS OF CONJUNCTIVITIS

Gram positive coccus	Staphylococcus streptococcus or pneumococcus
Gram negative coccus	Gonococcus or meningococcus
Gram positive bacillus	*C diphtherae*
Gram negative bacillus	*H influenzae H conjunctividitis* (Koch Weeks) *H duplex* (Morax Axenfeld) *P tularensis P pestis M mallei* or *B melitensis*
Carbolfuchsin stain (acid fast)	*M tuberculosis* or *M leprae*
Mononucleosis	Epidemic keratoconjunctivitis or Beal s conjunctivitis
Eosinophilia	Vernal conjunctivitis
Epithelial inclusion bodies	Trachoma or inclusion conjunctivitis
Darkfield microscopy	*T pallidum* and *T pertenue* (yaws)
Biopsy	Leptothrix or streptothrix

Equally effectual prophylaxis with lesser irritation can now be effected by 5 per cent sulfathiazole ointment warmed to a liquid consistency a microsuspension of sulfonamide crystals or a solution of penicillin containing 250 to 10 000 units to the cc

Precautions and Isolation—The patient with conjunctivitis must be regarded as an infectious menace He is required to take scrupulous care of hands Disposable tissues dressings napkins and handkerchiefs must be burned without delay Patients with *gonorrheal urethritis* are instructed as to personal hygiene The hands require scrubbing after touching the genitals and dressings are placed in an incinerator or are burned by a flame

Men and women with genital foci of infection are cautioned against the use of public swimming pools lest the contagion be transmitted to the eyes of others

In institutional life the child with conjunctivitis should be placed in strict *isolation* with special nursing care In factories or schools an outbreak of pink eye or virus conjunctivitis requires prompt and stringent action Afflicted individuals are barred from entrance into any public meeting place Tools and apparatus that are handled by many people are immersed in a 1 1000 solution of bichloride of mercury Each student or laborer is warned to scrub his hands thoroughly with soap and water after each fresh operation and on dismissal

Nonspecific Active Therapy —The most important nonspecific factor in the active treatment of acute conjunctivitis is *frequent lavage* of the conjunctival sac This is accomplished by the use of an *eye cup* or applications of *cold compresses* of physiological saline solution or 4 per cent boric acid

Specific Therapy —The indications for specific therapy depend upon the nature of the invading organism as detailed in the material which follows immediately Conjunctivitis may be treated locally or systemically Sulfonamides penicillin streptomycin, tyrothricin silver and zinc are available for topical application Concentration of the therapeutic agent may be increased by iontophoresis (p 1550), sulfonamide and penicillin may be introduced into the eye by direct injection under specialist supervision For specific systemic treatment the physician has the choice of sulfonamide and penicillin With the more serious and resistant infections all methods are combined

Staphylococcic Conjunctivitis —Conjunctivitis caused by the staphylococcus is very common and is usually observed as the *acute catarrhal type* of inflammation with a tendency to *chronicity* The causative organism may be demonstrated in smears and by culture The condition responds well to the generic treatment for conjunctivitis augmented by local applications of penicillin or 5 per cent *sulfathiazole ointment* Complications are rarely observed if treatment is continued for several weeks after acute symptoms have subsided

Streptococcic Conjunctivitis —Streptococcal conjunctivitis is seen relatively infrequently It may be manifested by an *acute catarrhal* or a *membranous* inflammatory process The bacteriological diagnosis is clearly apparent from the examination of smears and cultures The condition is managed in the manner of a staphylococcic invasion

Pneumococcic Conjunctivitis —Pneumococcic conjunctivitis is usually of the *acute catarrhal* variety and may be associated with *multiple subconjunctival hemorrhages* and *corneal ulceration* The pneumococcus is easily demonstrable in smears and cultures The condition responds promptly to local applications of *sulfonamide* or *tyrothricin* (30 mg per 100 cc) These agents have replaced the topical use of ethylhydrocupreine hydrochloride (optochin)

Meningococcic Conjunctivitis —Meningococcic conjunctivitis may occur in the course of a meningococcemia or as a purely local extra meningeal infection when meningococcal infections are rampant It appears as a hyperacute mucopurulent conjunctivitis indistinguishable from other more benign types It can be diagnosed by smears or scrapings when the organisms are exceedingly profuse

Meningococcal conjunctivitis responds to local sulfonamide therapy Internal administration of sulfonamides and injections of penicillin prevent generalized complications or the development of a carrier state

Gonorrheal Ophthalmia —Direct implant of the gonococcus into the conjunctival sac produces a *hyperacute type* of purulent conjunctivitis that may terminate in *blindness* After an incubation period of several hours to three days the lids appear swollen red and tender The conjunctiva has a bright velvety appearance pain is considerable and associated with a watery or slightly blood tinged discharge Organisms are demonstrable in epithelial scrapings but not in pus Constitutional symptoms such as fever malaise and chilliness may be present On or about the fourth or fifth days the swellings of lids and conjunctiva diminish discharge becomes frankly purulent and abundant and organisms are clearly demonstrable in smears and culture

In the era that preceded anti infective therapy complications were the rule in a gonorrheal conjunctivitis *Corneal ulcerations* (p 1626) were observed perforation often resulted in *panophthalmitis* and *loss of the eye* and healing was associated with *cicatricial deformities* and *corneal opacities* (p 1629)

Ophthalmia Neonatorum—The most hideous type of gonorrheal conjunctivitis is *ophthalmia neonatorum* which becomes apparent on about the second or third day after birth Both eyes show marked swelling of the lids chemosis purulent discharge and corneal ulceration Organisms are readily demonstrated by examination of smears and cultures Besides the gonococcus ophthalmia neonatorum may be due also to staphylococcal streptococcal and pneumococcal invasions or to an attack of inclusion blennorrhea (p 1623)

Treatment—The wide adoption of the Crede method of prophylaxis and therapy with sulfonamides and penicillin have removed most of the threat of gonorrheal ophthalmia *Prevention* is accomplished in the newborn by the immediate instillation of penicillin 5 per cent sulfathiazole ointment a microsuspension of sulfonamide crystals or 1 or 2 drops of 0.5 to 1 per cent silver nitrate *Active treatment* consists of local and systemic administrations of sulfonamide (p 88) or penicillin (p 106) The occasional presence of sulfonamide resistant strains and drug toxicity makes penicillin the preparation of choice

Influenzal Conjunctivitis —Influenzal conjunctivitis is usually of the *acute catarrhal variety* It is relatively benign and rarely accompanied by complications The organism may be demonstrated in smears and cultures Therapy with streptomycin may prove specific

Diphtheritic Conjunctivitis —The diphtheria bacillus is capable of producing a *membranous type* of conjunctivitis associated with a severe inflammatory reaction The diagnosis is established by demonstration of the organism in smears and cultures The presence of the condition requires the introduction of *diphtheria antitoxin* (p 310) in the manner of diphtheritic inflammations of the nasopharynx Local treatment is accomplished by palliative measures and application of penicillin ointment

Koch Weeks Conjunctivitis (Pink Eye)—The conjunctivitis that is caused by the Koch Weeks bacillus (*H conjunctivitidis*) occurs in *epidemic form* (Pink eye) There is an incubation period of thirty six hours and the attack usually lasts about two weeks Slim *gram negative rods* are

demonstrable in smears and cultures Excellent responses follow the generic treatment for acute conjunctivitis (p 1619) combined with the local use of 5 per cent *sulfathiazole ointment*

Morax Axenfeld Conjunctivitis (Angular Conjunctivitis) —The Morax Axenfeld type of conjunctivitis is usually of the *subacute catarrhal* variety The organism (*H duplex*) shows a *predilection for involvement* of inner and outer *canthi lid margins* and surrounding *skin* This type of angular conjunctivitis is benign and is associated with a stringy mucopurulent discharge that is never very abundant *An occasional corneal ulcer* complicates the conjunctival infection

The Morax Axenfeld bacillus is demonstrable in smears and cultures (p 1619) The organism apparently secretes a protein dissolving ferment which acts by macerating epithelium The application of solutions of 0 05 to 0 5 per cent *zinc sulfate* acts as a specific The efficacy of the preparation is due to inhibition of proteolytic ferment rather than any bactericidal action

Dysentery Conjunctivitis —Late in the course of bacillary dysentery (p 243) a simple *catarrhal conjunctivitis* may be observed The infection is obviously metastatic since organisms are not demonstrable by spread or culture Local therapy is attempted with sulfonamide since *secondary uveitis* (p 1632) sometimes develops within one or two weeks after the onset of the conjunctivitis

Tularemia Conjunctivitis —The *primary infection* of tularemia may be observed in the conjunctival sac Ordinarily the patient gives a history of tick bite or of handling the hide of a deer or a rabbit The *P tularensis* is demonstrable in smears or cultures (p 323) Specific antibiotic treatment with streptomycin is indicated (p 322)

Glanders Conjunctivitis —The primary lesion of glanders may occur in the lids or conjunctiva There is usually a clear history of handling horses *M mallei* may be grown on proper culture media (p 327) and is demonstrable in smears (p 1619) *Treatment* is symptomatic

B Granulosis Conjunctivitis —The bacillus granulosis of Noguchi may produce a *chronic follicular conjunctivitis* in children and young adults The infection is characterized by its mildness and the tendency of follicles to appear on the lower border of the lid The condition runs a benign course with complete restoration to normal Corneal complications and scarring are not observed *Treatment* is palliative and many ophthalmologists believe that the Noguchi organism is a secondary invader that appears in an *inclusion blennorrhea* (p 1623)

Syphilitic Conjunctivitis —The *primary lesion* of syphilis occasionally occurs in the conjunctival sac The clinical characteristics of the chancre are more often conspicuously absent The diagnosis is established once suspicion is aroused by *darkfield findings* (p 45)

A conjunctival chancre requires intensive systemic chemotherapy (p 340) using *arsenicals* or *penicillin*

Yaws Conjunctivitis —A granular conjunctivitis is often associated with yaws (p 351) With subsidence of the inflammation there may be *scarring* and *deformity* of the lid The diagnosis is made on demonstrating *T pertenue* by *darkfield examination* (p 45) *Treatment* is accomplished with the systemic use of penicillin or arsenicals (p 340)

Exanthematous Conjunctivitis —Exanthematous conjunctivitis is an acute catarrhal inflammation that occurs in *roseola* *rubeola* and *scarlet fever* In roseola the conjunctivitis precedes the cutaneous eruption and occurs simultaneously with Koplik spots (p 410) Palliative treatment is all that is required for the local lesion

Parinaud s Oculoglandular Syndrome —Parinaud s syndrome i character ized by unilocular granulomatous conjunctivitis and great enlargement of the pre auricular lymph glands Most cases appear to be due to a lepto thrix infection although the organisms of tularemia tuberculosis lympho pathia venereum and syphilis and yeasts have been shown to be etiolog ical agents Excision of the granuloma is indicated if the lesion fails to yield to ulfonamide and penicillin therapy

Inclusion Conjunctivitis (Inclusion Blennorrhea Swimming Pool Conjuncti viti)—Inclusion conjunctivitis is an acute conjunctivitis that is known as inclusion blennorrhea in the newborn and as swimming pool conjunc tivitis in the adult

Etiology —The condition is caused by a filtrable virus similar to that found in trachoma The virus is of genital origin and the reservoirs of infection are probably situated in male urethra and female cervix Epithe lial scrapings stained by the Giemsa technic reveal epithelial cell inclu sions and free elementary bodies 0 2 microns in diameter The disease has been reproduced by bacteria free suspensions of elementary bodies

Clinical Manifestations —The incubation period of inclusion conjunc tivitis is from 5 to 10 days The inflammatory process is most marked in the lower lids In the adult a severe acute follicular conjunctivitis with pre auricular gland enlargement occurs In infants due to absence of lymph follicles an intense papillary conjunctivitis with chemo is and swelling of the lids is encountered In both types a profuse mucopurulent discharge is present The cornea is never involved

Absence of gonococci in smears and the longer incubation period dif ferentiate inclusion blennorrhea from gonorrheal ophthalmia In the adult the involvement of the lower lid distinguishes it from trachoma

Course and Treatment —Until the advent of sulfonamide therapy in clusion conjunctivitis persisted in attenuated form for periods up to six months With local and systemic sulfonamide therapy inclu ion conjunc tivitis re ponds rapidly In many cases of inclusion blennorrhea local ther apy with 5 per cent sulfathiazole ointment is sufficient to produce cure in a week as lack of tearing in infants maintains a high concentration of the drug in the conjunctiva

Beal s Conjunctivitis —The acute follicular conjunctivitis of the type de cribed by Beal occurs in mild *epidemic form* It is characterized as an acute inflammation with edematous swelling more marked in the lower lid The bulbar conjunctiva is but slightly affected while the palpebral membrane especially in the lower fornices is studded with large follicles Considerable enlargement of pre auricular glands is always present

The diagnosis of Beal s conjunctivitis is suggested by the non purulent nature of the secretion and the demonstration of *mononuclear cells* in epithelial crapings Cultures are sterile or contain secondary invaders of little significance The etiological agent is probably a virus

Beal s conjunctivitis reaches its peak in three to ix days and then

subsides in one to three weeks The inflammatory process is self limited Corneal complications are not encountered and *symptomatic therapy* suffices Local instillations of 0 25 to 0.5 per cent silver nitrate and 0.25 to 1 per cent zinc sulfate may hasten healing

Vernal Conjunctivitis —See p 1650

Epidemic Keratoconjunctivitis —A virulent type of keratoconjunctivitis has been encountered *in epidemic form* The onset of the infection is an

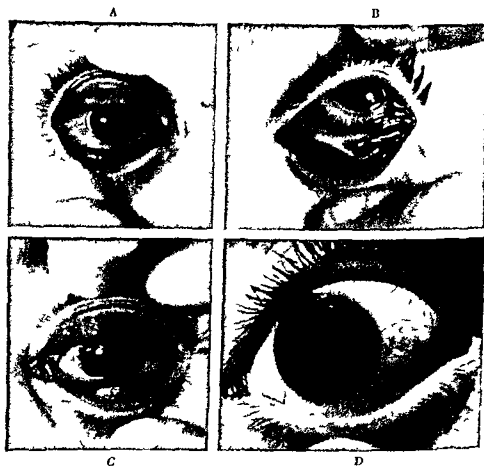


Fig 3 0—Epidemic keratoconjunctivitis *A* Note typical hyperemia and swelling of the palpebral and ocular conjunctiva The acutely inflamed appearance with absence of purulent discharge is characteristic of this supposedly virus infection *B* A pseudomembrane on the palpebral conjunctiva *C* Note the hyperemia glassy edema and *follicul tis of the palpebral* conjunctiva *D* Corneal opacities complicating epidemic keratoconjuncti itis Note the milky nebulae in the cornea *

nounced by varying symptoms and signs often identical with those of less intense varieties of acute conjunctivitis Hyperemia and swelling of the *palpebral conjunctiva* are noted The patient complains of irritation and a sandy *feeling suggesting the presence of a foreign body* Tearing may be intense After twelve to thirty six hours the *bulbar conjunctiva* becomes congested and hyperemic the lids appear edematous and a watery

* *Courtesy of Dr* Joseph G Molnar an l of Therapeutic Notes Parke D Co

secretion becomes manifest At this time scrapings reveal *mononuclear cells* leukocytes and bacterial organisms are conspicuously absent and cultures are sterile

After about a week the amount of lid edema may be so extreme that it is impossible to open the eyes A pseudomembranous inflammation is observed and if the cornea can be examined *subepithelial opacities* may be noted A dull *fronto occipital headache* is a relatively constant complaint *Pre auricular lymphadenopathy* is accompanied by swelling of *submaxillary and cervical nodes* The condition is differentiated from Beal s conjunctivitis (p 1623) by the presence of the corneal complications

The course of epidemic keratoconjunctivitis is protracted *Corneal* opacities remain long after the acute condition has subsided and may be permanent

Treatment—The present treatment of epidemic keratoconjunctivitis is purely symptomatic Specialist consultation is mandatory Injections of *convalescent serum* have been utilized with reported success *Sulfonamide*



Fig 8 1—Trachoma Early hypertrophic stage

and *penicillin therapy* is worthy of trial in view of the protracted course of the disease and its unfortunate aftermaths

Trachoma—Trachoma causes an appalling amount of suffering blindness and economic loss throughout the world The infection is overwhelmingly associated with poverty and lack of cleanliness It is almost universal among the natives along the eastern Mediterranean especially in Egypt Palestine Iraq and Persia It is very frequent in North Africa Russia Poland Hungary Japan China Mexico Northern Brazil and Argentina In the United States it is fairly common among native Americans living in a zone that reaches from the Alleghenies to Kansas and Oklahoma including eastern Kentucky Tennessee Virginia West Virginia southern Illinois southern Indiana and the Carolinas Ten per cent of the Indian population of the United States have trachoma but Negroes seem to have a definite immunity

Etiology—Trachoma is caused by a *filtrable virus* which can be seen microscopically in an early case as an epithelial cell *inclusion body* similar to that found in *inclusion conjunctivitis* (p 1623) The organisms are found in epithelial scrapings and occur intra and extra cellularly

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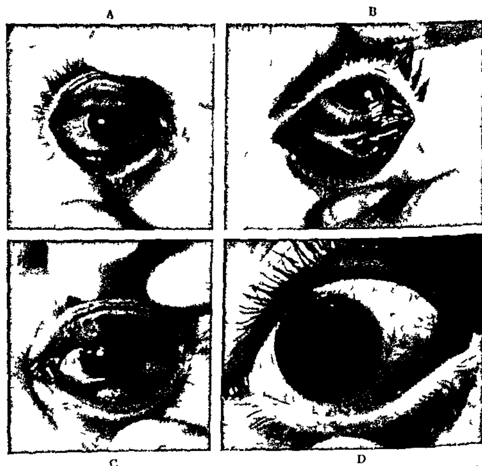


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* Courtesy of Dr Joseph G Molnar and of Therapeutic Notes Parke Davis & Co

temic toxic and infectious processes The lesions are usually chronic and resistant to treatment

Actinic keratitis a variant of punctate inflammation results from exposure to ultraviolet light and the glare of snow Klieg lights or an arc flash

Herpetic Keratitis—Vesicles of the cornea are seen in herpes simplex herpes zoster smallpox chickenpox and vaccinia They have been observed also in malaria typhoid fever following protein shock therapy and after treatment with diathermy

The usual corneal appearance is that of an acute superficial *vesicular keratitis* When the lesion assumes an arborescent appearance it is described as a *dendritic keratitis* Vesicular lesions of the cornea respond well to local applications of Tincture of Iodine and Glycerin

Corneal Ulcers—Ulceration of the cornea may result from local or systemic disturbances The diagnosis of the condition is aided by the green staining of the denuded area when *fluorescein* is locally applied

The normal corneal epithelium is an efficient barrier to the penetration of most micro organisms with the exception of the gonococcus and diphtheria bacillus When infiltrations and necrosis occur the ulcer may progress over the cornea (*serpent ulcer*) or it may penetrate deeper into the corneal stroma Damage to Bowman s membrane or stroma results in permanent *opacification*

Hypopyon Keratitis—When an extensive and penetrating corneal ulcer is sufficiently toxic the associated iridocyclitis results in an exudation from iris and ciliary body Pus collects in the anterior chamber (*hypopyon*) where it remains sterile unless corneal perforation occurs

Neuroparalytic Keratitis—After injury to the ophthalmic branch of the trigeminus the cornea becomes infiltrated and may ulcerate Trophic disturbances are prone to progress to perforation and loss of the eye unless the lid can be kept closed

Diffuse Parenchymatous or Interstitial Keratitis—Interstitial keratitis is an inflammation of the stroma of the cornea It is most often a manifestation of neonatal syphilis but it may also be encountered in tuberculosis focal infection leprosy trypanosomiasis and leishmaniasis In benign instances there may be almost complete clearing of the opacity though obliterated blood vessels remain permanently In more severe cases associated uveal tract inflammation and incomplete absorption of corneal opacities result in permanent and often serious impairment of vision

Keratitis Profunda—Keratitis profunda is an inflammation of the deeper layers of the cornea It occurs in adults and is associated with only moderately irritative symptoms Usually the disturbance clears within a few weeks The cause is usually unknown but it may follow trauma and has also been seen with upper respiratory infections and malaria

Keratitis Disciformis—In keratitis disciformis a disk shaped opacity is seen in the middle layers of the cornea The condition is of unknown etiology

Sclerosing Keratitis—In the course of a *scleritis* (p 1631) the cornea may become involved by a vascularized triangular shaped opacity with apex toward the center of the orbit This lesion may become very exten

Metabolic Disturbances and Poisoning

May be encountered with xerosis keratomalacia and avitaminoses In conjunction with Schuller Hand Christian disease and hyper thyroidism From exposure to carbon disulfide and chrysarobin

As Local Manifestation of Systemic Infection

May complicate tuberculosis leprosy syphilis yaws herpes simplex herpes zoster measles smallpox chickenpox vaccinia malaria trypanosomiasis and leishmaniasis May be due to focal infection

In Allergies

Particularly phlyctenular conjunctivitis

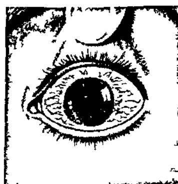
With Increased Intra ocular Tension

In glaucoma

Clinical Manifestations —Inflammation of the cornea is suspected when the patient complains of local pain photophobia lacrimation blepharo spasm and impairment of vision Examination reveals dulness of the cor



A



B

Fig 3?? —*A* Interstitial keratitis *B* Herpetic keratitis (dendritic ulcer)

nea and there appear scattered *dull grey infiltrations* or *spots* which stain green with fluorescein if the epithelium has been denuded Circumcorneal injection in the absence of discharge attests to an infection of deeper layers of the visual organs With keratitis there is always evidence of mild or severe *irritation of the iris* the *pupil* is miotic the iris may be clouded and exudate may be observed in the *anterior chamber* Upon even slight suspicion of corneal or uveal tract involvement the assistance of the ophthalmologist is sought

Superficial Keratitis —Superficial keratitis often accompanies various types of *conjunctivitis* (p 1616) and is also seen with *analogues of the dermatoses* (p 1564) A prominent feature of corneal involvements in *trachoma* is the appearance of the *pannus* (p 1625)

Superficial Punctate Keratitis —Superficial punctate keratitis is char acterized by *dot like superficial infiltrations* which stain with fluorescein Most commonly this condition is associated with chronic conjunctivitis but it may also be seen in avitaminosis allergy focal infection and sys

injections of nonspecific protein (p 1552) penicillin (p 106) or streptomycin (p 103) with the inauguration of hyperthermia (p 1552)

Transplantation of the cornea (p 1557) has been successfully practiced by expert surgeons Patients with opacities of the membrane are advised to consider this type of surgical procedure (keratoplasty)

EPISCLERITIS

The origins of episcleritis are similar to those of *keratitis* (p 1626) The condition is of less moment since it does not hold so serious a threat to vision

The onset of an episcleritis is announced by slight pain in the eye photophobia and lacrimation Examination reveals a dark red or purplish area in the ciliary region The lesion may be slightly raised and tender on pressure It is differentiated from *conjunctivitis* by the fact that instillation of epinephrine fails to cause any blanching The *investigation* of an episcleritis follows along the lines suggested in *keratitis* (p 1626) The



Fig 3 3—Scleritis



Fig 3 4—Acute iritis

commoner causes are *focal infection gout atrophic arthritis allergy tuberculosis* and *syphilis*

Treatment—*Local treatment* consists of the application of hot compresses and instillations of an atropine derivative to maintain dilatation of the pupil If there is considerable pain anesthetic drops and epinephrine (p 1548) give considerable alleviation of distress Empirically salicylates (p 383?) are administered orally in large doses Other *systemic measures* include elimination of foci of infection the use of an autogenous vaccine and desensitization with tuberculin

SCLERITIS

In scleritis there is an involvement of the entire thickness of the sclera The manifestations are those of an *episcleritis* (p 1631) except that the intensity is augmented more extensive areas are involved and the entire limbus may be surrounded by an annular process

Scleritis is usually associated with a *sclerosing keratitis* (p 1629) *secondary glaucomatous change* (p 1581) and involvement of the *uveal tract* (p 1632) After healing of the scleritis the weakened membrane may bulge producing an *ectasia*

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sive and interfere with vision after healing A tuberculous etiology is suspected

Keratitis e Lagophthalmo —Corneal inflammation may result from drying of the cornea due to incomplete closure of the lids following exophthalmos or facial nerve paralysis In milder forms it is prevented by the use of protective ointments on retiring Following malignant exophthalmos radical surgery is required to save the eyes

Diagnosis —The diagnosis of keratitis and its origin pose joint problems for ophthalmologist and practitioner The *ophthalmologist* is concerned with examination of the visual apparatus and the detection of local phenomena that stand in possible etiologic relationship to the corneal lesion The *practitioner* turns his attention to systemic manifestations whose significance requires interpretation

The recital of eating habits may suggest the possibility of an *avitaminosis* (p 616) Family and personal histories are significant in an *allergy* (p 547) Estimation of the *basal metabolic rate* assists in establishing the diagnosis of *hyperthyroidism* (p 1197) *Sensory examination* of the head and neck reveals other than corneal disturbances in *trigeminal lesions* (p 1481) *Tuberculin reactions* (p 262) and a *chest radiograph* are required with suspicion of involvement with the acid fast bacillus (p 252) and *serologic tests for syphilis* (p 337) are performed on blood and spinal fluid before definite commitment as to etiology

In the presence of a keratitis that is otherwise inexplicable the possible significance of *focal infection* (p 42) is investigated *Dental roentgenograms* are taken *vitality tests* are made and an opinion is required from the *dental surgeon* (p 1681) relative to possible foci that are not uncovered in such a survey Roentgenograms also are made of the *nasal accessory sinuses* but the fact that these reveal nothing of a positive nature should not deter the practitioner from referring his patient to the rhinologist for *local inspection* and *diagnostic lavage of the antrum* (p 2128)

Treatment —The *local treatment* of the keratitis is specialist province In all but mildest examples the pupil is kept dilated by atropine or a substitute (p 1548) Dark glasses are used to relieve photophobia and protect the membranes Hot compresses are beneficial and soothing in the vast majority of instances and the associated conjunctivitis is treated according to previously described methods (p 1619) *Adhesions between* cornea and conjunctiva are prevented by separating the lids with vaseline introduced at the end of a stirring rod

Systemic therapy in keratitis depends upon the fundamental underlying condition Syphilis requires antiluetic therapy which is instituted with great vigor Many ophthalmologists favor desensitization with tuberculin (p 1551) in keratitis of otherwise unknown etiology High vitamin diets and accessory vitamin feedings can do no harm if there is any *suspicion* of a deficiency Foci of infection are eradicated in the instance of culpable teeth and tonsils disease of the accessory nasal sinus may be overcome by lavage or appropriate surgery (p 2128) Smears and cultures are taken from focal areas of infection and pathogens are grown for skin testing and the manufacture of an autogenous vaccine (p 80)

Persistence of keratitis requires more heroic therapy in the nature of

nasal or postnasal discharge (p 2100) and radiopacities The rhinological survey is not complete without *diagnostic lavage of the antrum* and spreads and cultures of material for the detection of a possible pathogen With the completion of these examinations attention is directed to *male* and *female genitals* The *male urethra* (p 2340) is milked and the *prostate* is massaged the *female cervix* is inspected and the *adnexa* are examined bimanually (p 3468)

Uveitis also may accompany or follow obvious infections such as measles scarlet fever variola varicella typhoid fever cholera dysentery malaria rheumatic fever brucellosis influenza pertussis meningococcus meningitis meningococcemia mumps and leprosy

Clinical Manifestations —The clinical manifestations of uveitis vary according to the site of maximum involvement and the relative intensity of the process *Iritis* and *cyclitis* are discussed together as *iridocyclitis* but *choroiditis* is separately considered

Iridocyclitis —With iridocyclitis the patient complains of *pain diminution of vision photophobia* and *lacrimation* The pain in the eye may be most severe and may radiate to forehead temple or cheek Pain of this variety does not occur in *conjunctivitis* an important differential point in diagnosis (p 1618)

Local examination reveals *hyperemia* of the entire eyeball Injection is most marked around the cornea indicating that it is *ciliary* in type The pre auricular gland is not palpable the secretion appears watery and is not purulent The *anterior chamber* is clouded and fine particles or floaters are seen on slit lamp examination An abnormal ray or aqueous flare in the anterior chamber is observed by focal illumination Later *keratitic precipitates* (K P S) are seen The *pupil* is small its reaction is sluggish and it may be irregular if *synechias* have formed When cyclitis is severe there is marked swelling of the upper lid and chemosis and tenderness in the ciliary region intra ocular tension is decreased deposits on the anterior lens capsule are seen in the pupillary zone and the vitreous is hazy

COURSE —An *acute iritis* usually lasts three to six weeks and terminates favorably unless complications such as *secondary glaucoma* (p 1581) are encountered The *chronic types of iritis* are seemingly benign but they may be persistent and recurrent until finally the patient is left with a badly scarred eye complicated by *cataract* (p 1592) or a *glaucoma* (p 1578)

Acute iridocyclitis follows the same course as acute iritis but it is more severe and late results are more disturbing In addition to cataract formation *detachment of the retina* (p 1573) and *atrophy of the eyeball* may result

Chronic cyclitis or uveitis occurs most often in young adults Its onset is insidious and the condition may not be called to the attention of the physician until serious damage has occurred The usual presenting complaint is a *vitreous haze* or a diffuse dust like *speckling of the vitreous* which causes interference with vision

Choroiditis —The subjective symptoms of choroiditis are limited to *visual disturbances Pain* is conspicuously absent and the defect in vision escapes the patient s notice for a long period of time The herald manifestations are observations of flashes of light (*photopsia*) *vitreous opacities*

The presence of scleritis requires *specialist consultation* The investigation of the condition and treatment follow along the lines established for a *keratitis* (p 1626) Many ophthalmologists lay great stress upon the importance of tuberculosis as an etiologic factor Both scleritis and episcleritis occur in association with *atrophic arthritis* (p 2910) and other arthropathies

UVEITIS

The uveal tract is made up of iris ciliary body and choroid Inflammatory disturbances of components of the uveal tract are very similar and rarely occur in isolated form *Iritis* is usually associated with *cyclitis* cyclitis rarely exists without iritis and each produces or results from *anterior choroiditis*

Etiology—A wide variety of etiologic factors may result in the production of a uveitis Local causes include *analogues of the dermatoses* (p 1564) local trauma resulting in *sympathetic ophthalmia* (p 1569) and spread of *infections* from *conjunctiva* or *cornea* (p 1626)

Metabolic Causes—Much more often uveitis results from systemic metabolic or infectious mechanisms Considerable difference of opinion exists as to the relative importance of each factor Older writers were impressed with the *metabolic origins* of uveitis The ocular lesion was said to occur most often in the *gouty* the *rheumatic* with *intestinal autointoxication* in *nephritis* and in *diabetes mellitus* It is our opinion and experience that these factors are of minor significance

Infections—There is increasing belief that uveitis is the result of *systemic infection* but again a difference of opinion exists as to the relative importance and the frequency with which individual factors are participants Older clinicians were impressed with the significance of syphilis but this is negligible in present day practice Present opinion is divided as to the relationship between tuberculosis and focal infection One large group of ophthalmologists believes that *tuberculous uveitis* is exceedingly common It is their opinion that the uveal tract may be involved without significant tuberculosis elsewhere Their basis for the diagnosis of tuberculosis rests upon the results of the tuberculin reaction *therapy is* directed at desensitization with that substance (p 1551)

Our experiences favor the paramount importance of *focal infection* We are not greatly impressed by the tuberculosis theory nor do we have great faith in the protracted administration of tuberculin for purposes of desensitization It is our contention that an adequate search for foci of infection is rarely unsuccessful in the discovery of a possible nest Therapy is conducted in optimum manner by attention to the relationship between distant and local lesion

At risk of repetition the technic of seeking a focus of infection is again summarized Roentgenograms are taken of *teeth* and *nasal accessory sinuses* The *dental surgeon* is consulted for visual examination of the *gums vitality tests* and *peri apical explorations* of suspicious teeth for diagnostic purposes and to obtain material for bacteriologic smears and cultures The examination of the *tonsils* is scrupulous remembering that the most innocent appearing organ may have an abscess in its center The *rhinologist's* opinion of the nose and throat is essential particularly in the presence of gross abnormalities such as *nasal obstruction* (p 2732)

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metamorphopsia (distortion of images) *macropsia* (enlargement of images) *micropsia* (diminution of images) and *scotomas*

In acute choroiditis the ophthalmoscope reveals pale yellow or greyish white *fluffy areas* with ill defined edges Retinal vessels appear normal unless there is an associated *retinitis* (p 1637) After a variable time the exudate organizes fibrous tissue is formed and the choroid undergoes atrophy As a result of these changes the *white sclera* shines through and gives the lesion a dead white appearance *Pigment* is frequently deposited in or around the healed areas The presence of *vitreous opacities* suggests that there has been additional major involvement of the ciliary body

Choroiditis rarely occurs as an isolated lesion most often it is associated with *retinitis* Its complications include *uveitis* *optic neuritis* with secondary atrophy *cataract formation* and *sclerochoroiditis*

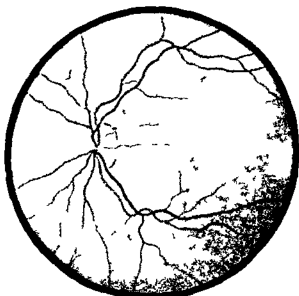


Fig 395 —Acute chorioretinitis *

DIFFUSE CHOROIDITIS —Diffuse choroiditis which is relatively rare consists of the coalescence of patches of exudate with involvement of the *greater part of the fundus* the retina appears edematous and cloudy eventually there is organization and ophthalmoscopy reveals white scarred areas over which the normally appearing retinal vessels course With *deposition of pigment* secondary pigmentary degeneration occurs which simulates the primary pigmentary degenerations of the retina (p 1592)

DISSEMINATED CHOROIDITIS —The disseminated type of choroiditis is said to be *syphilitic* (p 331) The entire choroid presents numerous round or irregular foci *Vitreous opacities* are present together with a certain amount of edema of the *optic disk* which later may become *atrophic* Massive choroiditis occurs in toxoplasmosis (p 535) particularly in newborn infants

* Gifford Textbook of Ophthalmology

CIRCUMSCRIBED EXUDATIVE CHOROIDITIS—In circumscribed exudative choroiditis one or more large foci are seen to be superficial or deep The *juxtapapillary choroiditis of Jensen* is an inflammation that is just adjacent to the disk It is associated with a sector shaped defect of the visual field thus differentiating the condition from *optic neuritis* (p 1641) or *congenital medullation of nerve fibers* (p 1563)

MACULAR OR CENTRAL CHOROIDITIS—Macular choroiditis involves the choroid in the region of the macula Since the retina is thin in this area there is considerable edema and elevation of the membrane and a *central scotoma* appears with marked *loss of vision*

Subacute and Chronic Uveitis—Subacute and chronic uveitis include *iritis iridocyclitis* and *choroiditis* In these inflammations there is limited destruction of tissue the lesion tends to remain focal exudation is slight and the cellular reaction is usually of the mononuclear variety *Exudates* appear as fine cellular *precipitates* on the posterior surface of the cornea Dust like *opacities* appear in the vitreous and abundant outpourings of large amounts of *fibrin* cause adhesions to bind the iris to the lens or form a cyclitic membrane behind the lens

The choroidal changes consist of exudation with mononuclear cellular reactions The end results of uveal inflammation are *atrophy of the eyeball secondary glaucoma cataract formation detachment of the retina optic atrophy* and *anterior or posterior synechiae* in which there are adhesions respectively to the cornea or lens

Suppurative Uveitis—Suppurative uveitis is usually of exogenous origin though it may occur endogenously in the course of an abscess or a generalized infection Localization of the suppurative process to the choroid or the posterior portion of the uvea gives rise to *endophthalmitis* terminating in the formation of an *abscess of the vitreous* The latter is suspected by the *yellowish reflex* to the interior of the eye Endophthalmitis may result in atrophy of the eyeball characterized by softening detachment of the retina and the formation of massive scar tissue in the vitreous With involvement of the *anterior uvea* the suppurative uveitis becomes more violent With involvement of all of the structures of the eyeball a *panophthalmitis* develops

The cardinal characteristics of suppurative uveitis include intense *pain* in the eyeball *protrusion* the formation of *pus* within the eyeball *perforation* and *phthisis bulbi* in which the eye shrinks after the perforation

Uveoparotid Fever (Heerfordt s)—Uveoparotid fever is an infection which is marked by chronic inflammation of parotid gland and uvea It is usually accompanied by chronic iridocyclitis unilateral facial paralysis lassitude and a subfebrile temperature reaction Its origin is unknown and treatment is symptomatic

Sarcoidosis—The syndrome of Boeck s sarcoidosis (p 3271) may be accompanied by conjunctivitis iritis and uveitis Roentgen therapy may be effective in expert hands

Diagnosis—The diagnosis of uveitis is of importance to the practitioner since the lesion requires immediate reference to the *consulting ophthalmologist* The distinctions between *acute conjunctivitis* and *acute glaucoma* are summarized in the chart on page 1618

Treatment—The treatment of uveitis requires complete cooperation be

tween ophthalmologist and practitioner Local therapy is the main concern of the former whereas the latter seeks the etiologic mechanism and attempts to deal with more fundamental disturbance

Local Treatment—As soon as the attending physician can be certain of the diagnosis of iridocyclitis even in the presence of an increase in intra ocular tension mydriatics are instilled locally to accomplish dilatation of the pupil rest for the eye and prevention of the formation of adhesions A 1 per cent solution of *atropine sulfate* is administered in drop doses three to six times daily depending upon the pupillary reaction If the patient or the eye appears sensitive to atropine 0 2 to 0.5 per cent *hyoscine hydrobromide* is substituted

Hot compresses are appled locally using saline solution or boric acid These simple applications probably accomplish as much as exposures to *infra-red ray or medical diathermy It is the custom of ophthalmologists* to use liberal doses of the *salicylates* If these have no specific action at least they act as an *analgesic Dark glasses* are prescribed and the patient is best placed in a darkened quiet room

Nonspecific Treatment—While the primary cause of the uveitis is being sought the process of healing may be stimulated by injections of *foreign protein* (p 1552) In the home it is safer to use intramuscular injections of 8 to 12 cc *boiled milk* In institutional practice intravenous *typhoid vaccine* may be employed (p 1552)

In the more severe types of uveitis with progression a trial with *anti infective agents* is worthy of consideration Oral doses of sulfadiazine are administered for a minimum period of three or four days with the usual precautions relative to the discovery of untoward manifestations particularly in the circulating blood (p 88) Concurrently or later intramuscular injections of penicillin may be given (p 106)

Specific Treatment—The most *important principles are* discovery of a possible focus of infection and prompt institution of remedial therapy It is our opinion that there is less danger of stirring up difficulty by an attack on the focus during the acute phases of the ophthalmological disorder *than in procrastinating with the hazard of permanent damage* to the visual apparatus Infected or even suspicious *teeth* are removed (p 1681) important or essential teeth may be managed by peri apical exploration and amputation *tonsils* are enucleated the *antrums* are irrigated and the sinuses are drained by intranasal or *extranasal surgery* (p 2128) *Smears* and *cultures* are taken from the focus a bacteria free filtrate is obtained for *skin testing immunization* is accomplished by means of an autogenous vaccine (p 80)

Those who favor the theory of the tuberculous origin of uveitis *are* less concerned with seeking and eliminating foci of infection and depend for therapy upon *desensitization with tuberculins* (p 1551) Probatory courses of antibiotic therapy using sulfonamides (p 88) penicillin (p 106) and streptomycin (p 103) merit trial when vision is seriously impaired

Patients with active syphilis receive *antiluetic therapy* (p 340) Generalized *ultraviolet radiation* (p 1550) has been suggested for the management of tuberculous uveitis With persistence of difficulty *roentgen therapy* may be tried

Surgery—The complication of a *secondary glaucoma* (p 1581) may

require the specialist to perform *paracentesis* of the eye A keratome is introduced into the anterior chamber to permit escape of aqueous fluid and the opening is maintained for a period of a week In persistent and recurrent iritis an *iridectomy* (p 1558) may be required At other times the *Lagrange operation* (p 1558) is performed and sclera is removed with iris

RETINITIS

Inflammation of the retina is usually secondary to *uveal infection* (p 1632) Ophthalmoscopy reveals clouding of retina The disk margin is congested and indistinct and exudates and hemorrhages are noted Later proliferative changes with deposition of masses of fibrous tissue result in distortion of the retina due to the formation of grey tumor like masses In final degenerative atrophy of the retina nerve elements disappear blood vessels appear changed and pigment is deposited

The patient with retinitis is referred to the consulting ophthalmologist Meantime efforts are made to reveal the more fundamental causative condition True inflammatory processes are seen in *syphilis* and *tuberculosis* Retinopathies are often associated with *arteriosclerosis* (p 976) *hypertension* (p 900) *toxemias of pregnancy* (p 2638) *diabetes mellitus* (p 1246) and *nephropathies* (p 2362) Petechiae are seen in bacteremias especially subacute bacterial endocarditis (p 1021)

Retinal Hemorrhages —Retinal hemorrhages may involve retina sub hyaloid areas or vitreous An end result of organization of the clot is seen in retinitis proliferans

Capillary Bleedings —Hemorrhages into the retina occur essentially from the capillaries The appearance of a hemorrhage in the retina depends on its situation When it occurs in the nerve fiber layer it is striate or flame shaped when situated deeper in the retina it is usually rounded or irregularly shaped The color is usually uniformly red but sometimes there are pale central areas While some retinal hemorrhages are rapidly absorbed and leave no trace of their presence others remain longer and are associated with scarring and white or yellow deposits Hemorrhages are usually asymptomatic but large bleedings situated near the macula may result in interference with vision

The *causes* of retinal hemorrhages include trauma disturbances of the local circulation such as obstruction in venous thrombosis papilledema and subarachnoid hemorrhage the vascular retinopathies of hypertension nephritis diabetes and arteriosclerosis local inflammatory disease of the retina toxic febrile infectious or cachectic states and diseases of the hematopoietic system such as the anemias leukemias purpuras and hemophilia

Before referring the patient with retinal hemorrhage to the ophthalmologist a careful systemic survey is required A *hemogram* may reveal hematopoietic disorders *renal* and *cardiac functional tests* are made *blood chemical analyses* and tests of *bleeding* (p 3706) and *coagulation* (p 3706) are performed In the presence of any striking abnormality the systemic condition is evaluated in reference to the local lesion Otherwise it is the responsibility of the specialist to discover the local etiologic mechanism

DIFFERENTIAL DIAGNOSIS OF

Reductions in Visual Acuity

Reduction in visual acuity may range from minor defects to total blindness The term amblyopia is employed to connote imperfect sensation of the retina without organic lesions involving the visual apparatus When there are demonstrable disturbances of the components that make up the apparatus for the detection of sight the word amaurosis is employed. Blindness embraces both amblyopia and amaurosis

	DIAGNOSTIC FEATURES
Psychogenic	Hysterical amblyopia Absence of organic findings
Disturbances of Ocular Adnexa	Conjunctivitis e pecially trachoma and ophthalmia neonatorum Dacryocystitis particularly in infancy Blinding filariasis
Disturbances of Fibrous Tunic	Keratitis scleritis and episcleritis with corneal ulcerations dystrophies and opacifications noted by slit lamp examination
Disturbances of Vascular Tunic	Choroiditis iritis iridocyclitis uveitis rupture of choroid and sympathetic ophthalmia Refer to speciali t for slit lamp examination and fundus observations
Disturbances of Chambers and Refractive Media of Eye	Cataract Glaucoma with increased intraocular tension Penetrating wounds and hemorrhages involving vitreous with danger of sympathetic ophthalmia
Disturbances of Neural Elements	Retinitis photoretinitis retinitis pigmentosa retinitis proliferans hemorrhage and detachment of retina optic neuritis papilledema senile degeneration of macula and retinoblastoma Refer to specialist for fundus examination
Disturbances of Blood Vessels of Eye	Obstruction of embolism of retinal arteries or veins Refer to specialist for confirmation and treatment
Disturbances of Orbit	Orbital cellulitis periostitis osteomyelitis and abscess Often secondary to inflammation of nasal accessory passages Refer to ophthalmologist and rhinologist Metastatic carcinoma and melanosarcoma Proptosis with exophthalmos
Errors of Refraction and Accommodation	Ametropia presbyopia and asthenopia Refer for refraction
Strabismus	With oculomotor trochlear and abducens paralyses With non paralytic strabismus and phorias Refer to ophthalmologist for diplopia tests
Hemic	Anemias leukemia and thrombocytopenic purpuras with characteristic hemograms.
Cardio-renal Disturbances	Hypertension, hypertensive toxemia of pregnancy and hypertensive encephalopathy associated with nephropathies Get renal function tests and check blood chemistry (p. 3712)

CONTINUED

Systemic Infection	Syphilis of optic nerve with positive serology Meningococcemia with positive blood culture Malaria with demonstrable parasites in blood smears Leprosy with anesthetic nodules Trypanosomiasis in epidemic areas Smallpox and herpes ophthalmicus with vesicular lesions involving cornea Focal infection with secondary uveitis
Contiguous Infection	Of nasal accessory sinuses especially sphenoethmoids Get x rays and refer to rhinologist for local examination and diagnostic lavage
Organic Neuropathies	Brain tumors abscesses cerebrovascular accidents and injuries involving optic pathways Supplement neurologic examination with x rays of skull and spinal fluid examinations Multiple sclerosis with disseminated lesions and pallor of temporal half of disk.
Metabolic Disturbances	Schüller Hand Christian disease in children with diabetes insipidus and osseous lesions Amaurotic family idiocy with cherry red spot at macula Avitaminosis A Uncontrolled acidosis
Poisonings	Alcohol particularly methyl Arsenic Quinine salicylate optochin aspidium, dinitrophenol (obesity cures) and tobacco Optic atrophy with alcohol and arsenic Cataract formation with dinitrophenol Amblyopia with quinine and salicylate

Preretinal (Subhyaloid) Hemorrhages—Subhyaloid hemorrhages have very characteristic appearances They usually occur near the posterior pole and are single and very large A dark red grapelike mass is seen which is circular and projects anteriorly into the vitreous This type of hemorrhage occurs particularly in *leukemia* (p 1100) and may be a herald manifestation of the condition Its appearance demands careful blood studies (p 3692)

Vitreous Hemorrhages—Any profuse retinal or preretinal hemorrhage may invade the vitreous body When this occurs the patient notes *impairment of vision* which may be complete A large hemorrhage abolishes the red reflex of the fundus and precludes ophthalmoscopic examinations of the interior of the eye Thus any sudden loss of vision in which fundus examination is impossible and in which nothing of the interior of the eye can be made out must be considered as due to a vitreous hemorrhage

The introduction of vitreous or spinal fluid may help absorption of vitreous hemorrhage

Retinitis Proliferans—When the blood clot following a vitreous hemorrhage becomes organized a retinitis proliferans develops The ophthalmoscopic picture consists of whitish connective tissue strands containing blood vessels which are derived from the retina and run deep into the vitreous There is serious *diminution of vision* and often ultimate *detachment of the retina* The condition is most often seen in uncontrolled *diabetes mellitus* (p 1246)

PERINEURITIS

Perineuritis (peripheral interstitial optic neuritis) is essentially a meningitis secondary to basal meningitis or suppurative pansinusitis It produces concentric contraction of the visual fields Treatment is directed at the primary lesion Syphilitic adhesive arachnoiditis requires specific therapy and operative interference by the ophthalmologist

OPTIC NEURITIS

Optic neuritis is an inflammation of the optic nerve (II) in any part of its course When it occurs behind the eyeball it is a *retrobulbar neuritis* when it occurs in the papilla it is a *papillitis* Optic neuritis is of frequent occurrence It arises from multiple etiologic factors and is of grave significance since it may terminate in loss of vision

Classification —Inflammations of the optic nerves may result from damage to *extraneural elements* of the media of the eye or to *neural factors* involving optic nerve optic chiasm optic tract primary visual end stations in the thalamus optic radiation or calcarine cortex

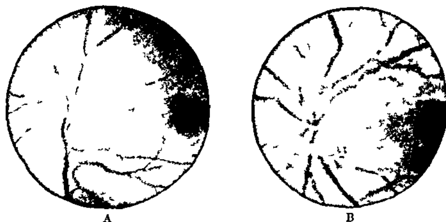


Fig 396 —*A* Postneuritic atrophy *B* Optic neuritis

Axial Optic Neuritis —Axial optic neuritis is usually of the retrobulbar variety The inflammation may be acute or chronic and occurs most often in young females The onset is announced by sudden cloudiness or dimness of vision frontal headache and orbital pain which is increased by pressure on or movement of the eyeballs

Examination reveals *mydriasis* and a *central visual defect* or *scotoma* The fundus appears hyperemic and there may be some blurring and swelling of the disk Progression of the lesion leads to *optic atrophy* with *temporal pallor* which may be unilateral or bilateral Retrobulbar neuritis is seen most often in multiple sclerosis (p 1504) and with nasal sinusitis (p 2124)

Leber's Atrophy —A hereditary axial neuritis affecting males and transmitted by unaffected females (Leber's atrophy) is characterized by an increasing *central scotoma* which affects *color vision* before it involves form *Nyctalopia* (*night blindness*) is a common symptom and total blindness occurs in almost one third of those who are affected The appearance of the fundus is that of an *optic atrophy* most marked temporally

Intra ocular Optic Neuritis or Papillitis —Papillitis is initiated by sudden and marked peripheral contraction of the fields of vision although there

* Gifford, Textbook of Ophthalmology

may be a central scotoma Impairment of vision may precede ophthalmoscopic findings which consist of redness and swelling of the disk blurring of disk margins dilatation of veins narrowing of arteries and sometimes hemorrhage and exudate Recovery may be complete but there is generally some degree of post neuritic or secondary atrophy marked by a dirty gray white appearance of the nerve head filling in of the physiological cup and sheathing of blood vessels (perivasculitis) Papillitis is often confused with *papilledema* (p 1577) and pseudoneuritis (p 1564) The marked loss of vision with sudden onset and a central scotoma generally distinguishes the condition from papilledema The degree of swelling is also less rarely exceeding 2 diopters *Pseudoneuritis* is differentiated by the absence of venous engorgement hemorrhages or exudates the normal sized blind spot and the fact that the condition is non progressive

Diffuse Optic Neuritis—Involvement of the entire optic nerve is often associated with a variety of *infections* which include influenza syphilis scarlet fever malaria yellow fever erysipelas and epidemic encephalitis The severity is such that the amblyopia is accompanied by a *papilledema* that may reach 4 or 5 diopters of swelling

Etiology—The causes of optic neuritis are as varied as the clinical manifestations They differ according to the site of the disturbance

- Extraneural Factors
  - Primary Retinitis
  - Retinitis Pigmentosa
  - Secondary Retinitis
    - Albuminuric
    - Syphilitic
    - Degenerative as in Amaurotic Family Idiocy
- Neural Factors
  - Peripheral due to local injury or disease involving the nerve within the orbit or as far back as the optic chiasm
    - Fracture of Base of Skull
    - Orbital Neoplasm
    - Neoplasm of Orbital Gyrus or Frontal Lobe
    - Gliomas of Optic Nerve
    - Neurofibromas of Optic Sheath
    - Periostitis of Walls of Optic Foramen
  - Orbital Cellulitis secondary to infections of eyelid or nasal accessory sinuses (sphenoid and ethmoid)
- The Optic Chiasm
  - Lesions of the Hypophysis
  - Involvement of the Sella Turcica
  - Sphenoid Sinusitis
- The Optic Tract
  - Cerebrovascular Disease
  - Neoplasms of the Base of the Skull
  - Localized Meningitis
- The Primary Visual End Station
  - Inflammations of lateral geniculate bodies or the pulvinar of the thalamus (vascular or neoplastic)
- The Optic Radiation
  - Vascular Disease Inflammation or Neoplasm of the Occipital Lobe
- The Calcarine Cortex
  - Degenerative Sclerotic Inflammatory Neoplastic or Vascular Disorders of the Occipital Lobes

Clinical Manifestations—The clinical manifestations of optic neuritis consist of subjective complaints evidences of defects in the visual field and abnormalities noted by ophthalmoscopic examination

Subjective Complaints—The patient may complain of clouding of vision frontal headaches orbital pain day blindness night blindness (nyctalopia), field defects, or visual perversions such as red vision (erythropsia) after cataract extraction and yellow vision (xanthopsia) after the excessive use of santonin (p 1896) or digitalis (p 860)

Visual Field Defects—In an *axial neuritis* the field defects are central with retention of peripheral vision Often the disturbance is first apparent with color rather than black and white By contrast there is a concentric field defect in the *intra ocular types of neuritis* with preserva

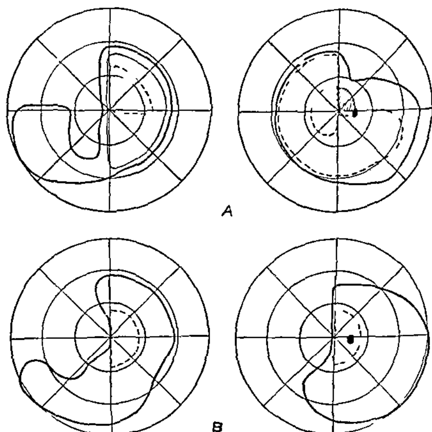


Fig 397—*A* Bitemporal hemianopia Different phases shown by quantitative testing *B* Homonymous hemianopia incongruous *

tion of central vision The more ominous *diffuse neuritis* causes a complete amblyopia or blindness

The visual disturbances serve also to localize the site of the defect from before backward *Unilateral blindness* follows destruction of a single peripheral area A lesion of the central portion of the optic chiasm produces loss of function in the nasal halves of both eyes (*bitemporal hemianopsia*) most often seen in hypophyseal neoplasms (p 1175) This lesion is known also as a *heteronymous hemianopsia* Lesions behind the chiasm produce a *homonymous hemianopsia* in which the field defect involves the

* Traquair in Berens The Eye and Its Diseases

ipsilateral nasal half and the *contralateral temporal portion* The production of a *binasal hemianopsia* requires the operation of two separate lesions involving the lateral portions of the chiasm and is all but unknown *Quadrantic hemianopsias* detectable only by tangent screen examinations arise with lesions of the temporal lobe involving temporal portions of the optic radiation the cuneate lobule above the calcarine fissure or the lingual gyrus

Ophthalmoscopy—Examination with the ophthalmoscope reveals redness blurring and swelling of the disk in an active peripheral neuritis The

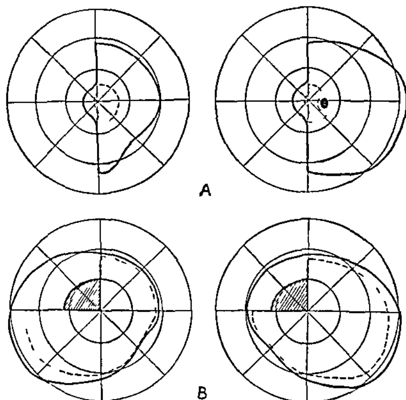


Fig 328—A Homonymous hemianopia congruous with macular sparing· enlargement of blind spot *B Homonymous hemianopic quadrant scotomas*

retrobulbar types are characterized by pallor particularly in the temporal halves as illustrated in multiple sclerosis tabes and poisonings with alcohol and tobacco With *secondary optic atrophies* the lamina cribrosa which normally is visible in the central cup disappears entirely as opposed to the appearance in the primary atrophy where it can be seen up to the edge of the disk (Fig 326)

Beside the changes in the disk varying degrees of *papilledema* may be observed even in the absence of increased intracranial pressure (p 1468) the veins are engorged the presence of *retinal hemorrhages* suggests vas

T aquair in Berens The Eye and Its D seases

tient recovered from the operation showed marked narrowing of a comparatively short segment of small intestine (Fig 513A and 513B) In addition to the narrowing there was a destruction of the normal mucosal markings in the involved segment with a number of small, rounded translucent areas The findings were persistently present during repeated observations of the small bowel At times the loop of small intestine proximal to the area of stenosis showed moderate dilatation At no time however was the dilatation very marked in spite of the fact that the degree of stenosis of the involved segment appeared to be considerable As noted in the biopsy examination, the pathologic process causing the abnormality in the roentgenogram was due to tuberculosis

A tuberculous lesion may produce obstruction of the small intestine



Fig 514 Tuberculous enteritis with obstruction of a loop of the jejunum

G N female aged 37 Six years previously the patient had had a resection of 15 inches of ileum for tuberculous enteritis

The report of macroscopic examination was as follows Specimen is a section of the ileum approximately 15 inches in length with a portion of the mesentery About 3 inches from one end there is an annular tumor mass about 1 inch in length firm and constricting the lumen On opening the intestine longitudinally, the mucosal surface is necrotic

and there is a small abscess in the wall containing light yellow pus The wall is thickened and firm About 3 inches further on there is a smaller annular growth, firm with necrotic mucosal surface About 3 inches from the caseation necrosis surrounded by endothelioid cells round cells and giant cells Similar tubercles are found in the mesenteric lymph nodes In one area of the intestine there is a large tuberculous abscess with giant cells



Fig 515 (A *Left*) Intestinal tuberculosis Appearance at 2 hours Note the alternating areas of narrowing and dilatation of the loops of small intestine The narrowed loops are irregular in outline and exhibit an abnormal mucosal structure (B *Right*) Same patient as is shown in (A) Appearance at 5 hours Essentially similar findings in the appearance of the loops of small intestine may be noted at this time (C *Center*) Same patient as is shown in (A) and (B) Appearance at 24 hours Note in particular the abnormal appearance of the cecum and ascending colon

opposite end there is a small infiltration in the wall about 1 5 x 1 5 cm The mesenteric nodes are numerous firm and large varying from 0 5 cm to 1 cm in diameter

The report of the microscopic examination was Sections show the mucosa to be ulcerated over large areas and the wall to be infiltrated by numerous round cells Throughout all sections are found numerous areas of round cells endothelioid cells and fibroblastic proliferation of the wall

The diagnosis was tuberculous enteritis and mesenteric lymphadenitis

About 1 year later the patient returned complaining of progressive abdominal swelling At operation the peritoneal cavity contained approximately 7 to 8 liters of clear yellowish fluid Both the parietal and visceral

peritoneum were studded with myriads of raised, small rounded, whitish tubercles The liver stomach spleen, both kidneys uterus *tubes and ovaries as well as the intestine were* covered with these tubercles

A biopsy was taken from the peritoneum Pathologic diagnosis was tuberculous peritonitis The patient continued to lose strength and a few years later returned complaining of cramplike abdominal pain vomiting, pro*gressive abdominal distention and increasing* constipation There was moderate weight loss Operation at this time showed the jejunum to be dilated about four times the normal size, the result of a constricting mass This was closely adherent to a large retroperitoneal circumscribed fluctuant mass An enteroenterostomy was done

The diagnosis was tuberculous enteritis with small bowel obstruction The patient gradually went downhill and died a few months later

Roentgen examination (Fig 514) revealed the marked dilatation of the jejunum as well as the area of constriction produced by the *tuberculous lesion*

Widespread involvement of the small intestine is illustrated by the next case

H A female, aged 24 Following the birth of her second child, 3 months before her admission to the hospital she complained of weakness, fatigue and 1 month later, of episodes of epigastric pain accompanied by diarrhea The diarrhea became progressively worse and on one occasion she stated that the *stool was black* She had lost considerable weight Cough was present

Physical examination of the abdomen was negative except for generalized tenderness Examination of the chest including roentgen studies showed evidence of pulmonary tuberculosis The sputum was positive for tubercle *bacilli While on the ward* she ran a temperature up to 104°F Examination of the larynx showed the vocal cords to be moderately edematous The diarrhea continued and she died on the fortieth day after admission



Fig 516 (A, *Left*) Intestinal tuberculosis Appearance at 3 hours (B, *Center*) Same patient as is shown in (A) Appearance at 4 hours (C *Right*) Same patient as is shown in (A) and (B) Appearance at 6 hours Note in particular the alternating areas of constriction and dilatation of the loops of the small intestine The narrowed portions are very irregular in outline The cecum and ascending colon are also irregularly narrowed and exhibit an abnormal mucosal appearance

Roentgen examination at *2* hours (Fig 515A) showed alternating areas of dilatation and constriction of the loops of small intes*tine, particularly of the ileum The constricted* areas were of irregular outline Some of the segments showed polypoid changes of the mucosa The appearance was that of a serious organic derangement of the small intestine to account for the diarrhea The persistence of these abnormalities was further confirmed in the 5 hour observation (Fig 515B) at which time it was noted that the cecum and the ascending colon were devoid of barium although the rest of the colon and the small bowel contained barium At 24 hours (Fig 515C) a small amount of barium was still present in the terminal ileum There was marked deformity of the cecum the ascend*ing colon and the right half of the transverse*

colon These roentgen findings in the presence of an active pulmonary tuberculosis, and a sputum positive for tubercle bacilli justified the diagnosis of tuberculosis of the small and large intestine

The changes in tuberculosis of the small bowel are shown in the following case

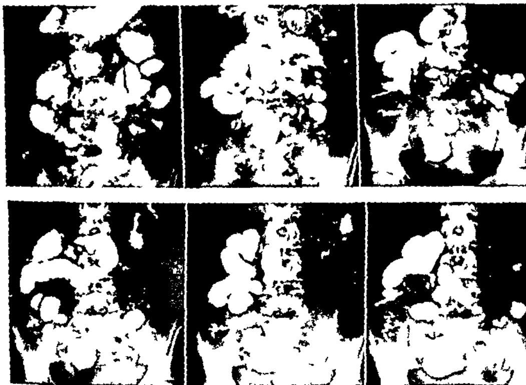


Fig 517 (A *Upper left*) Tuberculosis of the small intestine Appearance 1 hour after the ingestion of barium Note that the loops exhibit the segmentation dilatation and the ironing out of the contour similar to that noted in sprue (B *Upper center*) Same patient as is shown in (A) Appearance at 2 hours (C *Upper right*) Same patient as is shown in (A) and (B) Appearance at 4 hours (D *Lower left*) Same patient as is shown in (A C) Appearance at 5 hours (E *Lower center*) Same patient as is shown in (D) Appearance at 5 hours (F *Lower right*) Same patient as is shown in (E) Appearance at 7 hours Note that the abnormal appearance of the loops of the small intestine was a constant feature throughout this prolonged examination

A Y S N a Chinaman aged 49 The patient had been feeling weak during the preceding 4 years He entered the hospital because of abdominal pain of 8 weeks duration associated with diarrhea He had only a slight cough and very little sputum

Examination revealed tuberculosis of the lungs and of the epididymis The abdomen was distended Loops of intestine were seen moving beneath the abdominal wall The abdomen felt doughy Tenderness was most marked in the right lower quadrant The patient was febrile throughout his temperature reaching 104 F No sputum was available for study but *examination of the gastric* contents on two occasions revealed acid fast bacilli The course was progressively downhill and the patient died within a month of hospitalization

A roentgen study of the small bowel was made with hourly observations At 3 hours (Fig 516A) the loops were dilated segmented and the contour ironed out In one interconnecting area which was visualized the lumen was narrowed and the contour irregular At 4 hours (Fig 516B) the loops

peritoneum were studded with myriads of raised small, rounded, whitish tubercles The liver, stomach spleen, both kidneys, uterus, tubes and ovaries, as well as the intestine were covered with these tubercles

A biopsy was taken from the peritoneum *Pathologic* diagnosis was tuberculous peritonitis The patient continued to lose strength and a few years later returned complaining of cramplike abdominal pain, vomiting, progressive abdominal distention and increasing constipation There was moderate weight loss Operation at this time showed the jejunum to be dilated about four times the normal size, the result of a constricting mass This was closely adherent to a large retroperitoneal circumscribed fluctuant mass An enteroenterostomy was done

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are natural In the terminal ileum there are numerous erosions of the mucosa not surrounded by inflammation and not extending through the submucosa These erosions occur in little patches with normal mucosa in between, are not confluent measure up to 3 or 4 mm in diameter and do not run transversely At the ileocecal junction a sharp sudden change takes place The wall of the cecum is greatly thickened and the mucosa is extensively destroyed by a massive ulcerative lesion which involves the entire cecum but ends quite abruptly about 5 cm from the hepatic flexure The mucosal surface is dark red black in color and markedly hemorrhagic The remainder of the colon is grossly natural In the rectum the picture is similar to that seen in the cecum and again there is a very sharp line of demarcation at the level of the rectosigmoid junction The lesions extend through the entire rectum and involve even the anus The lymph nodes about the rectum and cecum are enlarged soft and caseous on section The mesentery of the small bowel contains a number of caseous nodes also

The diagnosis was ' tuberculous enteritis involving the ileum cecum and rectum Tuberculosis of mesenteric nodes

Roentgen examination (Fig 517) showed an unusual pattern of the small bowel There was evidence of (1) segmentation (2) moderate dilatation (3) an ironing out for the most part of the contour of the separated loop (4) Some of the intervening loops of small bowel appeared to be markedly constricted and of irregular outline (5) There was delayed motor function associated with narrowing of the extreme distal ileum



Fig 519 Ileocecal tuberculosis Note the concave deformity to either side of the ileocecal junction

That the findings were not of a transient nature and actually due to a profound derangement of the small intestine is emphasized by the fact that the abnormalities described were persistently present throughout the entire examination Because of the fact that the patient had pulmonary tuberculosis it was my opinion that the appearance of the small intestine was the result of

were again dilated and quite smooth except for one area which was moderately constricted and irregular in outline The most significant evidence of small bowel pathology was found in the 6 hour observation (Fig 516C) At this time some of the segments were considerably dilated and smooth in outline Other loops were markedly narrowed and of definitely irregular contour Of diagnostic importance also was the fact that the terminal ileum and cecum were narrowed and deformed The entire picture presented by these studies was that of serious organic disease of



FIG 518 Intestinal tuberculosis Note particularly the narrowed ileum with fistulous tracts also the narrowed cecum with small translucent areas within it

the small intestine as well as of the cecum in an individual with active pulmonary tuberculosis The diagnosis based on this evidence was intestinal tuberculosis

The roentgen appearance of the small intestine in tuberculosis may simulate that of sprue as illustrated by the following case which was confirmed by autopsy

H C female aged 25 The patient gave a 1 year history of weight loss progressive weakness and occasional profuse night sweats One month after the onset the patient complained of constant abdominal cramps and loose watery stools Bright blood was occasionally present in the stool

Physical examination was essentially negative except for the findings in the examination of the chest There were scattered rales in the left chest and diminished breath sounds at both bases Roentgenograms of the chest were interpreted as showing evidence of tuberculosis Examination of the sputum was negative for acidfast bacilli Proctoscopy showed ulceration and pinpoint bleeding areas in the rectum

The autopsy report was as follows There was about 500 cc of fluid in the right pleural space and about 500 cc in the left The left lung was densely adherent posteriorly and to the dome of the diaphragm About 30 cc of clear straw colored fluid was present in the pericardial cavity Examination of the lungs revealed pulmonary tuberculosis with early cavity formation The tracheobronchial lymph nodes showed evidence of tuberculosis There were tuberculous foci in the liver and ovaries

Findings for the gastro-intestinal tract were The esophagus stomach and jejunum

ileocecal junction may sometimes be noted in tuberculosis of this region

D P female aged 8 During the 7 months prior to her admission to the hospital the child had complained of abdominal pain and vomiting She had lost 15 pounds in weight Physical examination revealed a phlyctenular keratoconjunctivitis There was evidence of pulmonary tuberculosis Physical examination of the abdomen was negative except for generalized tenderness While under observation however further examination revealed a suspicious doughy feeling to the abdomen Her glomerate mass of retroperitoneal lymph nodes Scattered ragged irregular dirty grayish ulcerations were found in various portions of the small intestine Complete girdle like ulceration of the mucosa was present in some areas and healing and fibrosis in others There was thickening of the serosa beneath some of the ulcerations The cecum and the proximal portion of the ascending colon presented a deep red roughened granular appearance There were scattered ulcerations throughout the rest of the colon

Final diagnosis based on gross and microscopic examination was as follows 'Tuber

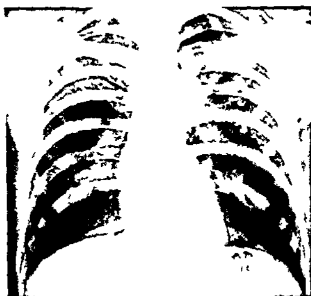


FIG 520 B Same patient as is shown in Figure 520 A Appearance of the chest

temperature while on the ward at times reached 105° and her pulse rate 140 She developed edema which was considered to be due to hypoproteinemia

The autopsy findings were There was tuberculosis of the right lung On opening the peritoneum there was about 100 cc of turbid yellowish fluid The omentum exhibited irregularly rounded yellowish firm nodules surrounded by narrow hemorrhagic zones Except for the proximal portion of the jejunum and the transverse descending and sigmoid colon the intestines were matted together in a mass of dense adhesions and yellow nodules

There were numerous nodules throughout the abdominal cavity There was a large conculous enteritis and colitis Ulceration and stenosis of the small intestine Tuberculous peritonitis Fibrocaseous tuberculosis of lymph nodes'

Roentgen examination of the cecum by means of a barium enema (Fig 519) showed an irregularly outlined concave deformity of the inner border of the cecum and a somewhat similar deformity of the outer or lateral border The entire cecum was narrowed A small amount of barium was present near the midportion of the inner border of the cecum apparently representing the ileocecal region

The deformity of the cecum, particularly in the presence of pulmonary tuberculosis justified the assumption that the lesion was tuberculous

an associated tuberculosis of the small intestine This view was confirmed by the autopsy findings as already described It is interesting to note that the general appearance of the small intestine in this case resembles that of sprue

Although there was some narrowing of the lumen of the ileum in several areas superimposed spasm was evidently an important factor in exaggerating the division of the bowel into the discrete segments seen in the roentgenograms



FIG 520 A Tuberculosis involving primarily the cecum and the ascending colon

The involved terminal ileum in tuberculosis may exhibit evidence of fistulous tracts

R S female aged 26 Six years before final hospitalization this patient was operated on and an embryonal carcinoma of the right ovary was removed Later she developed diarrhea, chills, fever and sweats and a productive cough There was progressive loss of weight and the patient died

The findings at autopsy were (1) fibrocaseous tuberculosis with ulceration of the cecum and colon (2) tuberculous peritonitis (3) miliary tuberculosis of the liver, spleen pleura and lungs (4) fatty degeneration of the liver In addition there was a mass of embryonal carcinoma in the retroperitoneal tissue invading the vena cava There were thrombi in the iliac veins

The detailed findings at autopsy of the gastro intestinal tract are of importance to an understanding of the roentgen findings

"The stomach contains a considerable amount of brownish fluid of watery consistency The small intestine shows no lesions until several small ulcers are found in the terminal ileum The ileocecal valve and cecum show great thickening of their walls and contain numerous fine nodular masses The mucosa shows no evident primary tumor but shows a great amount of necrosis The ileum and the cecum open into a sinus tract which is in the approximate position of the appendix, and the three lumina contain thick grumous, black, liquid material This sinus tract extends to the peritoneal surface of the right cornu of the uterus There are at least 30 ulcers in the large intestine and they extend from the cecum to the rectum The ones in the ascending colon are rather smaller about 3 mm across on the average while the pelvic colon contains in addition to the smaller ones eight to ten which are up to 1 cm across The ulcers are fairly well punched out, and are red in color No tuberclelike masses are found on the edges"

Roentgen examination had been made about 4 years before this patient s death At that time a study of the ileocecal region was made by the ingested meal method (Fig 518) In addition to marked narrowing of the terminal ileum, several sinus tracts were visible Proximal to this narrowed region the ileum was dilated and exhibited small translucent areas within it Another loop of terminal small intestine was also frayed and of irregularly narrowed contour However, it was not nearly so constricted as the most distal portion of the terminal ileum The cecum was narrowed, with irregularity in the contour of the lower pole and a mottled appearance throughout produced by small translucent areas within it The irregular contour of the inner border of the cecum at the lower pole was apparently due to thickening of the ileocecal valve, as found at autopsy The numerous small translucent areas throughout the cecum apparently corresponded to the many fine, nodular masses as discovered at autopsy Also sinus tract formation was verified at autopsy

A concave deformity of the inner border of the cecum proximal and distal to the

J C male aged 23 The patient gave a 4 year history of hoarseness and productive cough He later developed evidence of tuberculosis of the larynx and the lungs Three years later he developed abdominal cramps and watery nonbloody diarrhea The sputum was positive for tubercle bacilli at this time, and roentgen examination showed evidence of a bilateral pulmonary tuberculosis

Physical examination of the abdomen except for a right lower quadrant scar was essentially negative

Operation revealed a massive tuberculous involvement of the terminal ileum cecum ascending colon and hepatic flexure There was marked stenosis at the ileocecal valve and at the hepatic flexure The involved area was resected

The report of the macroscopic examination was Throughout the mucosa of the ileum there are many rounded raised nodules about 2 mm in diameter There are 2 small irregular ulcers near the ileocecal valve measuring 0 5 cm in diameter The cecum and first part of the ascending colon are narrowed The wall is thickened and fibrous in consistency The mucosa is absent except for occasional polyplike tags

The final diagnosis was 'Tuberculosis of the ileum, cecum and ascending colon with ulceration and fibrosis Tuberculosis of lymph node mesenteric

Roentgen examination by barium enema revealed considerable deformity with marked narrowing of the cecum the ascending colon and the hepatic flexure (Fig 521A) The aerogram (Fig 521B) also showed the strictured appearance of the involved segment of the proximal colon The stenosis was particularly marked in the ascending colon and the hepatic flexure The diagnosis of tuberculosis was made as a result of the extent of the lesion and the involvement of the cecum the ascending colon and the hepatic flexure in the presence of active pulmonary tuberculosis



Fig 522 A Tuberculosis of the small and the large intestine (ingested meal method)

Diffuse involvement of the entire cecum and ascending colon in intestinal tuberculosis is illustrated by the following case

G P, male, aged 26 This patient was operated on after an acute attack of right lower quadrant pain The surgeon reported a ruptured appendiceal abscess The patient was hospitalized for 21 days The incision continued to drain pus A fecal fistula developed This was traced as communicating with the cecum Examination of the chest showed the presence of pulmonary tuberculosis The sputum was positive for tubercle bacilli Except for one short period there was no diarrhea

At the time of reoperation for the fecal fistula, a piece of tissue was removed which was reported as showing evidence of tuberculosis

Roentgen examination showed a marked irregular narrowing involving the entire cecum and ascending colon (Fig 520A)

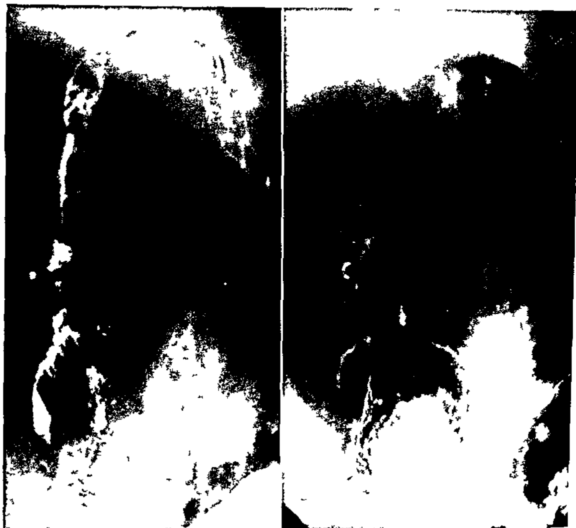


Fig 521 (A Left) Ileocecal tuberculosis (B Right) Same patient as is shown in (A) Aerogram showing strictured appearance of the involved bowel

Figure 520B shows the appearance of the lungs in this case

The roentgenologic appearance of the cecum and the ascending colon in conjunction with definite evidence of pulmonary tuberculosis justified the diagnosis of tuberculosis of the involved colon As noted above this was corroborated pathologically

The narrowing of the cecum and ascending colon may sometimes be very marked

scribed inspection of the mucosa of the terminal ileum reveals numerous slightly raised gray nodules averaging 5 mm in diameter with a depressed and in some cases reddened and roughened central crater An nular lesions constrict the lumen The mucosal surfaces in these regions are rough and gray at the periphery and rough and red in the central part of the band Numerous small nodules are found especially in the peripheral region of the annular lesions The mucosal surface of the cecum is rough dirty gray and red and nodular There are numerous mesenteric lymph nodes draining the in volved regions varying from 3 to 25 mm in size

The report of the microscopic examination was Sections from ileum and colon show tuberculous ulcers penetrating into the sub serosa The ulcers are lined by granulation tissue rich in plasma cells and lymphocytes and containing thrombosed and recanalized vessels There are caseous tubercles in the walls of the ulcers in the adjacent intestinal wall and in the intestinal and mesenteric lymphoid tissue *The process is principally* caseous with little fibrosis and rare giant cells

Roentgen examination by the ingested meal method (Fig 522A) showed areas of seg mentation and moderate dilatation of distal loops of ileum associated with narrowing and irregularity of contour Some of the moder ately dilated loops also showed superficial ir regularity of contour *The last few inches of* the terminal ileum exhibited irregularity of contour and an area of moderate narrowing in this loop about 1 inch proximal to its junction with the cecum The ileocecal area was wide and gaping The cecum and ascending colon and hepatic flexure showed a marked degree of distortion with narrowing irregularity of contour and destruction of the normal mu cosal *markings In the midportion of the* transverse colon there was an area of nar rowing with irregularity of contour and de struction of the normal mucosal markings

Examination by barium enema (Fig 522B) showed irregularity of the terminal ileum and cecum The colon then appeared normal except for an area of narrowing in the left half of the transverse colon The evidence then was *that of organic disease of the distal* ileum the cecum and the left half of the transverse colon in an individual with pul monary tuberculosis This evidence justified the opinion that the lesions were tuberculous

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Tuberculous involvement of the intestine may be characterized by skip areas

S M, male aged 38 Nineteen months before his admission to the hospital, the patient noted epigastric pain occurring about 5 to 6 times a day, and not relieved by soda He lost 30 pounds in the following 6 months and about that time developed a diarrhea with from 3 to 4 stools a day, never watery or bloody A number of months later he developed a chronic productive cough which was not purulent or bloody At the time of admission to the hospital his temperature was 101 4 pulse 142 respiration 21 He was extremely pale dyspneic and cyanotic There was considerable tenderness in the right lower quadrant Roentgen examination of the chest showed the presence of pulmonary tuberculosis and tuberculous pleural effusion

Autopsy disclosed fibrocaseous tuberculosis of the lungs with cavitation The peritoneal surface of the lower ileum, caecum and transverse bowel is marked by the presence of several annular, slightly contracted rough and dull red areas approximately 4 cm in breadth and bordered on either side by a lighter colored dirty grayish, rough area about 1 cm in breadth There are *2 such areas in the* terminal portion of the ileum, one about 18 inches, the other 9 inches from the ileocecal junction A similar area is present at the terminus of the ileum and blends into a thick rough gray and nodular cecum This cecal pathology extends up about 5 cm *towards the* ascending colon On the left side of the transverse bowel an annular lesion similar to those described in the ileum, is seen The mesentery supporting the pathological regions is thickened and contains numerous firm nodules, varying from 0 5 to 1 5 cm in diameter Especially about the cecal area is the peritoneum and mesentery thus thickened



Fig 522 B Same patient as is shown in Figure 522 A Examination after a barium enema Note particularly the involvement of the ileocecal region and of the left half of the transverse colon

In addition *to the annular lesions de*

scribed inspection of the mucosa of the terminal ileum reveals numerous slightly raised gray nodules averaging 5 mm in diameter with a depressed and in some cases reddened and roughened central crater An nular lesions constrict the lumen The mucosal surfaces in these regions are rough and gray at the periphery and rough and red in the central part of the band Numerous small nodules are found especially in the peripheral region of the annular lesions The mucosal surface of the cecum is rough dirty gray and red and nodular There are numerous mesenteric lymph nodes draining the in volved regions varying from 3 to 25 mm in size'

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54

Rectal Stricture Secondary to Lymphogranuloma Venereum

INCIDENCE AND PATHOLOGY

Lymphogranuloma venereum may be defined as a specific venereal disease caused by a filtrable virus and characterized by a transitory and insignificant primary lesion on the genitalia. It is associated with a subacute or chronic regional lymphadenopathy and, in some cases, rectal stricture is an important complication. The disease is not a new one, and buboes which resemble the lesions of lymphogranuloma venereum, were described by the ancient Greeks and Romans as well as by physicians, like John Hunter[1] in the latter part of the eighteenth and in the early nineteenth centuries. Some even described an herpetic like primary lesion, although the etiologic agent remained nebulous and was ascribed to malaria, tuberculosis, venereal infection (syphilis chancroid gonorrhea), scrofula and climatic conditions.

Nelaton,[2] who was among the first to distinguish this condition as a distinct clinical entity, gave a good description of the adenopathy and described cases with herpetic lesions on the penis. He thought it was due to an infection with a gential portal of entry. Marion and Candy[3] gave a complete pathologic and clinical description, but it was in 1913 that Durand, Nicolas and Favre[4] gave impetus to more widespread knowledge of the disease and named it "lymphogranuloma inguinale."

In 1925, Frei[5] introduced his skin reaction for testing the presence of the disease. Following the introduction of the Frei test there was a marked renewal of interest in lymphogranuloma inguinale and reports on the clinical and experimental aspects of the affection have been numerous. A vast literature has accumulated which was reviewed in a recent excellent monograph by Stannus citing over 900 references. In America, although isolated reports appeared in the literature, it was brought more clearly to the attention of the general medical profession by the publications of De Wolf and Van Cleve,[6] Sulzberger and Wise,[7] Cole[8] and Bloom.[9]

Descriptions of nonmalignant or inflammatory stricture of the rectum can be recognized in the medical literature of the past hundred years. It is in recent years, however, that the disease has emerged as an important sequela of lymphogranuloma venereum.

It is quite possible that the type of rectal stricture now known to be secondary to lymphogranuloma venereum may have been referred to by Jonathan Hutchinson[10] when he wrote:

> Amongst the local diseases which appear to be in some indirect way, connected with tertiary spyphilis we have non malignant strictures of the rectum. This form of stricture is much more common in women than in men, and it is the general testimony of observers that a large majority of those who suffer from it have at some former period had syphilis. ... Two difficulties present themselves: first, in a certain number of cases there is no reason to suspect syphilis and yet the disease is, for the most part, of the same character; and secondly, in the cases in which there is a conclusive history,

we can seldom do any good by constitutional treatment

The disease is not uncommon De Wolf and Van Cleve in 193? and Cole in 1933 reported 110 cases from one hospital covering a period of 3 years and Lichenstein who collected 154 cases over a period of 4 months estimated the number of cases applying to New Orleans Charity Hospital as well over 400 cases a year

From 1935 to 1938 187 cases of the disease were treated on the dermatologic service at Bellevue Hospital[11]

Bloom[1] reported upon his personal observation of over 100 cases of lymphogranuloma venereum at Bellevue The primary lesion is usually evanescent and herpetiform In some cases however, it may persist as an ulceration for weeks or months The chancre of lymphogranuloma venereum may frequently be intraurethral Following infection an inguinal adenitis may develop which may be transient or a bubo may develop having the characteristics of a firm indurated tumor attached to the underlying tissues However in only 1 of 41 cases of rectal stricture was there a typical inguinal bubo Bloom believed therefore that involvement of the inguinal nodes may protect the individual from the penetration of the virus into the pelvis Such inguinal involvement however does not preclude the development of rectal invasion as in pederasty, or the virus may spread directly by way of lymph vessels into the pelvic nodes Only a proctitis or colitis may develop in some case Involvement of the pelvic nodes may lead to rectal stricture Sequelae of lymphogranuloma venereum are due mainly to stasis as a result of occlusion of the lymphatics in turn due to destruction of the lymph nodes Because of destruction of the anorectal nodes there is stasis in the wall of the rectum with secondary ulceration elephantiasis and fistulous tract formation

Inflammatory and infectious changes are usually associated with rectal stricture and are responsible for the mucopurulent and sanguinopurulent discharge from the rectum Polypoid structures resembling hemorrhoids are commonly seen about the anus Fistulae in ano are frequently seen with rectal stricture They may be multiple and open upon the perianal and perineal regions They may be traced into the rectum several centimeters above the anus They may heal spontaneously but tend to recur in spite of all therapy even excision Rectovaginal fistulas with the passage of feces by way of the vagina are not uncommon

The symptoms of rectal stricture are quite characteristic Due to the reduction in size of the lumen of the rectum and its rigidity patients develop chronic increasing constipation and a change in caliber of the stools which may become ribbonlike This is associated with rectal tenesmus and pain The associated proctitis is evidenced by a viscid mucopurulent and sometimes sanguineous discharge more marked while straining at stool There may be alternating periods of diarrhea either due to the underlying proctitis or to drastic purges taken to overcome the obstipation

Strictures of long duration may be associated with marked cachexia weakness and anemia which may simulate a malignancy of the rectum

Proctoscopy is a valuable confirmatory procedure and permits biopsy and visual inspection of the stricture and the rectum below

ROENTGEN DIAGNOSIS

The following are the important roentgen findings in rectal stricture secondary to lymphogranuloma venereum

1 The process is diffuse in this type of rectal stricture and in every case that I have seen roentgenologically there has been narrowing of the anorectal region Thus in one case where roentgenologic examination showed the anorectal margin to be uninvolved and the narrowing to begin about 1 inch proximal to the anus the narrowing of the rectum was diagnosed as being due to malignancy although the Frei

test was positive The mass was resected and microscopic study showed definite evidence of carcinoma

While a failure to involve the entire rectum in the narrowing process speaks more in favor of malignancy, the reverse is not true That is to say, if the entire rectum is involved, the process may still be due to malignant infiltration

2 The narrowing is usually irregularly tubular and diffuse, without any very abrupt delimitation from the normal contour In malignancy there is more likely to be an abrupt sharply delimited termination of the lesion However, there may be exceptions to this so that this difference cannot be used as an absolute criterion for differentiating a malignant from a benign infiltrative process A finger printing type of deformity within the strictured region would favor the diagnosis of malignancy

3 The presence of fistulous tract formation, extending from the strictured area into the perirectal tissues, occurs not infrequently in cases of benign stricture due to lymphogranuloma venereum Roentgenologically, numerous fistulous tracts extending for various distances may be noted I have never seen such fistulous tracts in a case of malignancy of the rectum This evidence therefore, when present, is pathognomonic of the benign nature of the infiltrative process and is the safest criterion for differential diagnosis on a roentgenologic basis



Fig 523 (A *Left*) Rectal stricture secondary to lymphogranuloma venereum (B, *Right*) Same patient as is shown in (A) Appearance after employing the double contrast method of examination

Illustrative Cases The following are examples of the roentgen manifestations of rectal strictures secondary to lymphogranuloma venereum

Tubular narrowing of the entire anorectal area in lymphogranuloma venereum is illustrated by the first case

M V, aged 42 This patient had always been very constipated, particularly during the past 17 years The stools were very hard and of small diameter and accompanied by blood

Rectal examination revealed an annular stricture situated 5 cm above the external sphincter The stricture admitted only the tip of the index finger It felt fibrous External hemorrhoids were also present There was no evidence of inguinal adenopathy The Frei test was positive

Roentgen examination revealed the presence of a marked narrowing of the entire rectum and distal portion of the sigmoid with destruction of the normal mucosal markings (Fig 523A) The extent of the involvement, the destruction of the normal mucosal markings as well as the lack of distensibility of the involved region were well shown following the employment of the double contrast procedure with air injection (Fig 523B)

The appearance of fistulous tracts associated with the rectal stricture is illustrated by the next case

L F aged 38 This patient gave a 7 year history of progressive constipation pain and bloody stools A colostomy was performed for stricture of the rectum Later there was obstruction at the colostomy requiring further operative intervention

Roentgen examination of the rectum and sigmoid showed marked irregular tubular narrowing (Fig 524) None of the opaque substance (lipiodol) could be forced beyond the sigmoid into the descending colon In addition to the marked tubular narrowing a number of fistulous tracts were well shown extending into the perirectal tissues

In some cases the strictured area may be diffuse and involve a good part of the sigmoid as well as the entire rectum An example of this kind is illustrated by the next case





Fig 524 (*Left*) Rectal stricture secondary to lymphogranuloma venereum showing fistulous tracts into perirectal tissue

Fig 525 (*Right*) Stricture secondary to lymphogranuloma venereum involving the rectum and a part of the sigmoid Note the fistulas at the anorectal region

R H female aged 65 This patient said that she had always been constipated but that during the past year this had become much worse For several years she had noticed a purulent bloody discharge from the rectum which had gradually become more marked

The Frei test was positive Physical examination revealed the following

1 In the rectum there were cutaneous tabs about the anus irregular and slightly tender The rectal digital examination showed a stricture about 2 cm from the anus, but the tip of the finger could easily be passed through

test was positive The mass was resected and microscopic study showed definite evidence of carcinoma

While a failure to involve the entire rectum in the narrowing process speaks more in favor of malignancy, the reverse is not true That is to say, if the entire rectum is involved, the process may still be due to malignant infiltration

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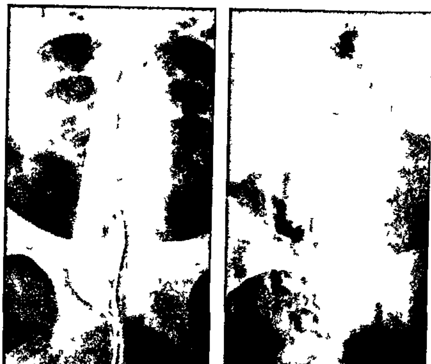


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rectum of irregular contour, may however be produced by a malignant infiltration

F O aged 60 The main complaint of this patient was increasing constipation with lower abdominal pain and rectal bleeding Digitally some 5 cm from the anal margin there was a firm annular mass surrounding the lumen of the lower bowel apparently fixed at the anterior wall of the rectum The tumor tissue projected into the lumen of the bowel The edges were irregular and nodular There was a stricture of the rectum due to the mass

Biopsy reveal d an adenocarcinoma of the rectum A colostomy was performed

Roentgen examination (Fig 527) revealed a diffu e irregularity with narrowing involving the entire rectum The fingerprinting defects were more suggestive of a new growth than a stricture of inflammatory origin

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this region The walls were very irregular The anal sphincter was relaxed

2 The inguinal nodes were slightly enlarged principally in the left groin freely movable and not tender

A specimen removed from the rectum and examined microscopically showed evidence of acute and chronic inflammation The tissue consisted of a small bit of smooth mucosal and vesiculous tissue containing many infiltrating lymphocytes and monocytes There was a mass of necrotic tissue with some red blood cells intermingled

The diagnosis was acute and chronic inflammation

Roentgen examination (Fig 525) revealed the presence of a diffuse narrowing of the entire rectum and sigmoid with superficial irregularity of contour There were several fine lines of barium at the anorectal margin strongly suggestive of fistulas

That absence of roentgenologic evidence of involvement of the anorectal junction in the process of tubular narrowing may be important in differential diagnosis is illustrated by the next case

J J, aged 63 For the past year this patient had been complaining of diarrhea with about 8 bowel movements a day Gross blood had been present in the stool during the past 6 months Rectal examination revealed a hard annular mass encircling the bowel The Frei test was positive The Wassermann test was strongly positive

At operation there was a carcinomatous mass involving the rectosigmoid this was annular, firm and about 2½ inches in extent The mass could be felt above the level of the pelvic peritoneum in the pouch of Douglas Numerous diverticula of the sigmoid colon were noted Microscopic examination revealed a colloid carcinoma

Roentgen examination (Fig 526) revealed a marked annular narrowing for a distance of about 2 inches with an essentially smooth outline but tubular in appearance The distal portion of the rectum appeared to be intact In spite of the positive Frei test and the strongly positive Wassermann, a diagnosis of malignancy was made roentgenologically because of the fact that the anorectal region appeared free of involvement

A diffuse tubular narrowing of the entire





Fig 526 (*Left*) Note the absence of tubular narrowing of the anorectal region although the rest of the rectum shows such narrowing The Frei test was positive Pathologically this was a carcinoma

Fig 527 (*Right*) Carcinoma of the rectum simulating the tubular narrowing of a benign stricture

rounding the diverticulum may result in tumefaction and secondary stenosis by contraction (peridiverticulitis) Apparently in some cases this is due to a thinning of the wall of the diverticulum so that micro organisms may readily penetrate through to the peritoneum with the production of a granulomatous hyperplasia As a result of the marked peridiverticulitis with thickening of the wall the stenosis may produce an appearance simulating that of carcinoma Even after resection the gross features may

Although diverticula are most often noted in the sigmoid every portion of the colon is vulnerable including the cecum Rarely only a solitary diverticulum may be present limited to the cecum As with diverticula generally it may enclose a fecalith, or be the site of serious inflammatory disease (Jackson[6] Cooke Pereira[8] and Jonas[9])

ROENTGEN DIAGNOSIS

Ordinarily the barium enema may be employed for the visualization of diverticula



FIG 528 Diverticula of the colon

appear like those of a malignancy, and only microscopic study may lead to a correct interpretation The presence of associated diverticula may suggest the true nature of the lesion The association of carcinoma and diverticulitis in the same region is not common and when this does occur may be considered as purely coincidental and not etiologic

An important complication of diverticula of the colon is fistulous tract formation with neighboring viscera particularly between the sigmoid and urinary bladder

of the colon However a failure to demonstrate diverticula by this method is not conclusive evidence of their absence The following additional procedures may be employed

1 In some cases when the colon is completely filled with barium diverticula actually present may be obscured unless they spring directly from the inner and outer borders In such cases a film taken after evacuation of most of the barium will succeed in bringing them into clearer relief

2 Even when diverticula are not demon

55

Diverticula of the Colon

ETIOLOGY

Cruveilhier[1] and Virchow[2] laid the groundwork for the anatomic pathologic description of diverticula of the colon

Diverticula of the colon are frequently present at the mesenteric border and correspond to areas of penetration of vessels through the muscularis, large vascular gaps in the intestinal wall leading to a decreased resistance of the intestine at these regions with herniation of intestinal mucosa through them The blood vessels often appear to guide the passage of the mucosa through the layers of the intestine Such areas of predilection might yield to even moderate intra intestinal pressure such as occurs in constipation or gaseous distention

To demonstrate the vulnerability of such areas of the colon to diverticulum formation Hansemann,[3] after excising a portion of the intestine distended it with water He thereby produced small bulges of varying sizes along the mesenteric border Graser[4] injected the inferior mesenteric artery of the body of a 37 year old woman and hardened the descending colon sigmoid flexure and rectum in alcohol He then noted that in some places the circular muscle was interrupted and the longitudinal muscle thinned so that, in these regions there was a hernial bulging of the mucosa which microscopically gave the appearance of false diverticula These bulgings were present where the larger vessels passed through the musculature

Such a relationship of the origin of diverticula of the colon to the sites at which the bowel is perforated by mesenteric vessels, however, does not always hold Diverticula may be present, not only at the mesenteric attachment, but also at other parts of its circumference, and are not infrequently found to grow into the appendices epiploicae To explain this fact Beer[5] predicated the assumption of the presence of localized areas of muscular weakness in regions of the intestine other than at the pathways of blood vessels at the mesenteric border

The most likely explanation of the etiology of diverticula of the colon is that they are due to pulsion as a result of the increased pressure within the bowel from the accumulation of feces or gas The fact that they are most apt to occur in the sigmoid suggests intracolonic pressure as a factor, since fecal stagnation and hardening of the stool are apt to be greatest in this region The diverticula then develop as herniations at sites of diminished resistance either along the mesenteric border where it is perforated by blood vessels, or elsewhere in the colon due to localized areas of diminished resistance in its wall

The diverticula appear as small pouches extending outward from the lumen of the bowel The narrow neck may be partially occluded by overlapping mucosa and may be localized only after a careful search When the diverticula extend into the appendices epiploicae it may be impossible to see them on external examination The diverticula are often occluded by fecal content within them This may become hardened, and the concretions cause perforation with abscess formation More frequent than perforation is inflammation due to mechanical irritation A hyperplastic process sur



Fig 530 Diverticula of the sigmoid



Fig 531 Diverticula of the sigmoid

strable in either the immediate or post evacuation films following the barium enema examination by the ingested meal method may succeed in filling the diverticula Films taken at 24, and if necessary at 48 hours may show the presence of diverticula otherwise not demonstrable, apparently due to the fact that the barium within the colon has a greater opportunity by virtue of its longer stay of seeping through the narrow necks and entering the diverticula

A prediverticular state in the formation sufficient size to be roentgenologically demonstrable The phenomenon of bowel contour may be a secondary result of their presence rather than the groundwork upon which they develop Moreover, as the result of marked narrowing of the necks of the diverticula, which may be further exaggerated by edema resulting from inflammation, it may be impossible to force barium into them Consequently, diverticula even though present may escape detection roentgenologically Only the effect upon the appearance and behavior of the bowel may be noted



Fig 529 Diverticula of the cecum and the ascending colon

of diverticula in which the normal haustral configuration of the colon is replaced by a ragged contour was described by Spriggs and Marxer[10] They claim to have noted the eventual development of diverticula in such areas of irregularity of bowel configuration with inflammatory processes finally leading to diverticulitis

Regarding *the attempt however to trace* the natural history of the development of diverticula by the roentgen method the possibility must be considered, in my opinion, *that in the stage of irregularity* of contour of the bowel and evidence of irritation the diverticula may have already been present, even though they have not assumed

The roentgen appearance of diverticula will vary with their size the narrowness of the neck, which may prevent complete filling and also the amount of fecal material *within the diverticulum itself Because of* partial blockage by a fecalith, the barium may outline the diverticulum in the form of a crescent For the stage free of clinical *symptoms the name diverticulosis'* is applied as opposed to the condition associated with clinical activity with inflammation and swelling of the mucosa and evidence of rigidity and tenderness over the area of involvement, called *diverticulitis* "

In a study of the mucosal relief of the colon in diverticulosis, the convergence of

the folds may be noted to the neck of the diverticulum without actually entering the sac itself Insufflation may show the diverticula in a clear and isolated manner against the background of the injected air In the absence of inflammation the colon retains its distensibility and the mucosal relief exhibits *a normal appearance In the presence* of the actual inflammation of diverticulitis there is a disorientation of the mucosal pattern the lumen may be narrowed and the configuration irregularly serrated





Fig 534 (A *Left*) Fecalith within a diverticulum of the cecum (B *Right*) Same patient *as is shown in* (A) Roentgen appearance of the fecalith after removal

In the presence of tumefaction as the result of chronic inflammation the differential diagnosis from new growth may be difficult or impossible The demonstration *of diverticula within the zone of the irreg*ularly outlined stenosed area is evidence in favor of a granuloma due to such diverticula The association of such a lesion with actual malignant infiltration must also be kept in mind

Illustrative Cases Figure 528 shows almost the entire *transverse colon containing* diverticula as well as the descending colon and sigmoid While many are rounded and globular in appearance others appear club-shaped Some are multilocular Examples of this latter type are particularly noticeable along the lower border of the transverse colon The cecum itself appears to be free of diverticula

That diverticula may also be present in the cecum and ascending colon is demon*strated by Figure 529 This is the post* evacuation film after a barium enema There is a residuum of barium retained in the diverticula although the cecum and ascending colon are otherwise practically free of barium

Variations in the size shape and contour of diverticula may be noted in Figure 530 and Figure 531 Incomplete filling of the diverticula *with barium is due to the fact* that fecal masses of various sizes are present within them This also explains why a diverticulum of the colon may be present and not be roentgenologically discernible at all the fecal content producing complete occlusion Also when the neck of the diver*ticulum is occluded because of inflamma*tion and edema none of the barium may be able to enter At times the number of diverticula may be enormous (Fig 532)



FIG 532 Diverticula of the sigmoid



FIG 533 Isolated diverticulum at the lateral border of the cecum

translucent apparently because of retained fecal content A few of the diverticula are almost completely filled with barium

Inflammatory tumefaction of the sigmoid secondary to diverticulitis has been previously described in the discussion of granulomas of the colon (Fig 444)

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An isolated diverticulum may be present in the cecum Usually when it occurs it is *located at the inner border Rarely, it may originate at the lateral border (Fig 533)*

A fecalith within a diverticulum may become sufficiently opaque to be recognizable in the roentgenogram This is illustrated by the following case

J P, aged 37 During the preceding 4 years, the patient had had intermittent at*tacks of severe pain in the right upper* quadrant which occasionally radiated into the right lower quadrant There was occasional vomiting No blood was present in the vomitus or stools There was a weight loss of 12 pounds

Physical examination of the abdomen was essentially negative except for a right lower quadrant scar (appendectomy 16 years before)

At operation a hard freely movable mass measuring 2½ to 3 cm in diameter was felt in the right lower quadrant apparently attached by a pedicle This pedicle proved to be the narrowed lumen of a diverticulum originating from the upper end of the cecum The remainder of the abdomen was negative The diverticulum was resected On opening the diverticulum a fecalith was *present*

Roentgen examination (Fig 534A) showed an irregularly rounded opaque area in the right lower quadrant Figure 534B represents the appearance of the fecalith

The mucosal appearance of the colon in relation to diverticula is shown in Figure 535 The mucosal folds stop abruptly at the diverticula Some of the diverticula are *rounded and filled with barium* others are translucent with a thin rim of barium, or cup shaped as a result of fecal material within them





Fig 535 (*Left*) Mucosal relief appearance in diverticula of the sigmoid
Fig 536 (*Right*) Diverticula of the colon Appearance after combined injection of barium and air

In addition to the appearance of diverticula as observed in relation to the mucosal relief of the colon they may also be studied by the double contrast method This is illustrated in Figure 536 While some of the diverticula may be seen extending *from the* lateral border, most of them are seen en face The rounded margin is demonstrable through a coating of barium The center is

HERNIATION AND EVENTRATION OF THE DIAPHRAGM

56

Herniation of the Diaphragm

ANATOMY OF DIAPHRAGM
ETIOLOGY OF DIAPHRAGMATIC HERNIA
EARLY DESCRIPTIONS
INCIDENCE AND TYPES
ROENTGEN DIAGNOSIS
ABSENCE OF THE DIAPHRAGM

ANATOMY OF DIAPHRAGM

The diaphragm is a musculofibrous septum of elliptical shape It is composed of a peripheral muscular portion and of a central tendon Anteriorly it is attached to the inner surface of the lower 6 or 7 ribs posteriorly, it is attached to the internal and external arcuate ligaments In addition the diaphragm is attached centrally to the lumbar vertebrae by its crura To either side of its attachment to the ensiform cartilage there is a deficiency in the muscle structure which is occupied by areolar tissue and lined by pleura above and abdominal peritoneum below These areas are known as the foramina of Morgagni Posteriorly there are similar areas of muscular deficiency between the fibers of the internal and external arcuate ligaments (foramina of Bochdalek)

The main openings in the diaphragm are for the aorta the esophagus and the vena cava In addition there are smaller apertures for the passage of the splanchnic nerves and the vena azygos minor

It is only in the mammalia that one finds complete development of a diaphragm In lower animals only the pericardial cavity *is separated from the abdominal cavity* Even in these cases a pericardioperitoneal *canal may be present* connecting the two cavities

*During embryonic life there is a com*munication between the pericardial pleural and peritoneal cavities A mesodermal structure called by His the septum transversum is the anlage from which the diaphragm develops In the 2 mm embryo the septum transversum is in the cervical region (Mall) There is then a gradual distal migration toward the lumbar region the septum carrying with it the phrenic nerve During early embryonic life the diaphragm is composed only of muscle Later the central portion atrophies with the development of a tendon

ETIOLOGY OF DIAPHRAGMATIC HERNIA

The following factors are important in considering the etiology of diaphragmatic hernia During the developmental process there may be a failure of closure of the pleuroperitoneal membrane perhaps due to a retardation of growth or a halt in normal development Abnormal development of the diaphragmatic muscle may lead to defects through which herniation may take place

The aortic hiatus has never been the location of a hernia Hernias occur anteriorly through the foramina of Morgagni (parasternal hernias) probably the result of increased intra abdominal pressure and may occasionally be bilateral Other diaphrag*matic hernias may occur centrally or pos*teriorly The latter result from a failure of closure of the pleuroperitoneal canals They therefore originate between the vertebral *and costal portions of the diaphragm in the* lumbar region

*The esophageal opening for the develop*ment of herniation results from a failure

lodged in the diaphragm and in the chest In order better to examine this condition without displacing the parts I opened the chest and found these parts without any coating *They had passed through an opening between* the fibres of what is called the nervous center of the diaphragm Although the hernia was very old the viscera had not developed any adhesions either to themselves or to the edge *of the aperture in the diaphragm* This circumstance accounted for their free mobility between the chest and abdomen

As regards the opening in the diaphragm it was oblong and one inch wide by two inches greater portion of the stomach had entered the chest and had carried along the entire omentum

The patient had for a long time been subject to attacks of what he believed was asthma and which probably had no other cause than the hernia since he felt relief as soon as he ate This patient died from an abdominal inflammation and I performed the autopsy Since those who thought that he was asthmatic had diagnosed this inflammation of the abdomen as thoracic hydropsy I began the autopsy by opening the chest There I found only an insignificant amount of fluid together



Fig 537 (A *Left*) Esophageal hiatus hernia (B *Right*) Same patient as is shown in (A) Mucosal appearance of the herniated portion of the stomach

long There was no hernial sac which was astounding for one might have expected a peritoneal sac but both the pleura and peritoneum were ruptured and united at the edge of the opening so that this opening had a natural appearance

The second hernia seems to me to be attributed to the same defect as the former This hernia was on the left side as in the former case and my colleagues have all assured me that the hernias which they have *seen were on the same side This leads me to* believe that the convex border of the liver protects the right side of the diaphragm It is also possible that the viscera on the left side are able to exert a greater pressure on the left diaphragm However that may be the second hernia was much larger than the first A with a tumor This tumor on the left side was as large at its base as its midline and ended in a soft cone of a height of three to four inches On exerting pressure over this tumor I felt some resistance the reason being that the entire abdomen expanded by the inflammation opposed itself to the displacement of the tumor The tumor however became somewhat diminished as I compressed it only to recur as soon as the compression ceased In order to palpate it in its entire extent *I had to separate it from the lung to* which it was attached by thick lymph On opening the abdomen I found the stomach the colon and the omentum in a condition which I have previously described The whole *abdomen was inflamed The portions included* in the abdomen were glued to one another

of the muscle of the diaphragm completely to surround the esophagus There is thus an area made up of peritoneum, pleura and interstitial tissue, forming a relaxed membrane, which is less resistant to the occurrence of herniation as the result of increased intra abdominal pressure

An abnormal development of the omental bursa *may lead to a herniation through the esophageal hiatus of congenital origin* This is due to a persistence of the infracardiac bursa which communicates with the omental bursa and therefore with the abdominal cavity There may result, therefore a direct communication through the diaphragm alongside the esophagus Through such a congenital opening a herniation of the stomach may develop

Rarer locations for herniation are the openings of the sympathetic nerve chains *and the opening of the vena azygos minor* Keith[1] reported herniations in the floor of the pericardium (pericardial hernia) so that the heart was surrounded by coils of small intestine

A unilateral lack of the diaphragm arises as a result of a failure of development of the pleuroperitoneal membrane The first definite case of absence of the left diaphragm proved by operation was reported by Harris and Clayton Greene[2] Richards[3] referred to one case of a monstrosity in *which there was a total lack of the entire* diaphragm

The greater frequency of hernias on the left side is apparently due to the protection afforded by the liver on the right side

EARLY DESCRIPTIONS

The first description of a congenital hernia of the diaphragm is to be found in the work of Riverius[4]

The son of Monsieur Rat a citizen of Montpellier, was twenty four years old and of a melancholy temperament, all summer long he had been tormented by an inveterate tertian fever, sometimes single and sometimes double Indeed in the month of September although he no longer suffered paroxysms of fever, his health was not of the best A certain quack had persuaded him to take antimony (perhaps improperly prepared) which stimulated him to very violent——though unproductive——attempts to vomit As a result these *efforts constantly wracked his frame* and reduced him to the last extremity in seven to eight hours causing his death toward evening The cadaver being opened the stomach was found to be in the right part of the thorax and to be filled with various fluids Doubtless this praeternatural situation prevented the stomach from dilating or turning over sufficiently to cause proper vomiting, since the ribs surrounded and pressed upon the stomach *Now the proof that this transposition* was congenital lay in the absence of the lung in this part of the thorax, and just one lobe upon the left side It is cause for wonder that this young man was not greatly oppressed by a difficulty in breathing throughout the course of his life and that prior to the above mentioned illness his body had duly performed all its functions for he had long been in the army, and he had contracted these fevers in the war

In his posthumous classic 'Traite des Maladies Chirurgicales' published in 1790 Petit[5] gave the following description of hernia of the diaphragm

I have seen 2 cases of hernia *of the dia*phragm and several of my colleagues have told me that they have also seen some cases The fibres of this muscle (the diaphragm) are not always so well knit together as not to permit separation in some instances in one or another place Under such condition it does not take considerable effort to push the abdominal viscera between these separate fibres and to force them into the chest Of the 2 thoracic *hernias which I have seen* 1 was *very old* at least according to the patient, who told us, some time before his death that for forty years of his life he was attacked from time to time by what he called 'stomach colic' He told me that with such an attack he had great difficulty in breathing, of such severity that *he thought he was about to die Also*, that no sooner these smothering attacks were over he felt nauseous, had to vomit although he did not vomit very much These attacks were accompanied by very intense pain He also stated that the alleged colic never attacked him when his stomach was filled and that he *could stop it by taking food* When this man died I performed the autopsy, at which I found a large portion of the colon, great omentum, and of the fundus of the stomach

and indeed that the diaphragm had been dilacerated in the circular nerve Almost no lung was found on the perforated left side since beyond doubt the lung had been destroyed by pus a slender shred remained attached to the ribs The entire stomach [had] ascended into the left side of the thorax and the heart with its capsule [pericardium] [had] moved over from its site to the right side where the patient when alive



FIG 539 (A *Left*) Herniation of the stomach Examination in the right oblique position Note the serrated appearance of the esophagus The termination of the esophagus is not visible (B *Right*) Same patient as is shown in (A) Examination in the left oblique position Note the displacement of the distal end of the esophagus by the herniated portion of the stomach

was destined to observe his cardiac pulse The heart was atrophied with an abundance of *citrine* water in the capsule [pericardium] The omentum and the pancreas were also putrefied almost all around the stomach

In this case history what certainly stands out above its other distinctive features is that the stomach changed its location But now there is no more leisure for writing Goodby to Your Excellency Given at Wittenberg this fifth day of May 1608

Additional early reports of diaphragmatic hernia are to be found in the publications of Astley Cooper[8] Morgagni,[9] Cruveilhier[10] Ballantyne[11] and Keith[1]

INCIDENCE AND TYPES

Diaphragmatic hernia is more common than is indicated by clinical and roentgenologic diagnosis Harrington[13] examined the diaphragm in the course of operations for other abdominal conditions and occasionally found small hernias that had not been suspected He recommended the division of hernias of the diaphragm into the nontraumatic and traumatic according to the following classification

A nontraumatic hernia may be congenital or acquired

1 If congenital the hernia is attributable to embryologic deficiency and usually

and the whole glued to the internal surface of the sac containing them I say expressly glued because there were no solid adhesions such as I have mentioned in other types of hernias

When the viscera were pulled from the sac, they kept sticking together in such a manner as to present the form of a tumor As regards the sac, I put my hand in and even turned it inside out I realize that it was nothing but a prolongation of the peritoneum, diaphragm and pleura together, without any rupture of the membrane or separation of the muscular and tendinous fibres of the diaphragm



FIG 538 Hernia of the stomach through the esophageal hiatus

Ambroise Pare[6] in 1575 published the first description of a hernia of the diaphragm of traumatic origin

The signs which indicate that the diaphragm is wounded are discomfort at the site of the wound, delirium that is to say disturbance of thought there is great difficulty in breathing, coughing and acute pain As the result of deep inspiration it sometimes occurs that the stomach and intestines are drawn into the thoracic cavity through the wound, as I have observed in two persons One was a stonemason s assistant who sustained a wound in the center of the diaphragm, of which he died on the third day Having opened the abdomen, I could not find the stomach, which amazed me greatly, thinking that it was a monstrous thing to be without a stomach But having diligently thought about the matter I finally learned that it had ascended into the thorax I noted that the wound in the diaphragm was just large enough to receive the thumb and having opened the thorax, I found the stomach inflated and filled with gas and a little fluid content

I do not want to omit the story of what happened a short time ago to a captain, who received a gunshot wound which entered at the end of the sternum near the ensiform cartilage, passing through the muscular part of the diaphragm and making its exit between the fifth and sixth ribs on the left side There persisted ever since a sort of colic because of which he always ate sparingly At autopsy there was found in the thoracic cavity a large portion of the colon filled with gas which had entered through the aperture in the diaphragm produced by the wound

A description of the next case of hernia of the diaphragm of traumatic origin is to be found in the work of Fabricius Hildanus,[7] in the form of a letter written to him under the date of May 1608

Furthermore while I do not doubt that you have had many rare and strange cases under your surgical care and have studiously written them up I thought that you might not object to hearing of the following case through me It was described by my father in law, Andrew Schato, of pious memory, formerly a professor of medicine in this Academy It runs as follows Heinricus Buscherhovius, 'Revaliensis Livonus' [place name], was tormented by melancholia and various attacks What finally happened to him was that, on returning to Wittenberg in Saxony, he was right murderously run through in front of the gate of the city on September 20 1562 The sword had been driven into the thorax below the fourth rib and had emerged from the back below the ninth rib Nevertheless, this wound healed inside of two months It actually happened that several months later, he was again a prey to absurd thoughts and he fell ill a little while after (on April 28 1583) On the following days he took (as nourishment) first water and food of all kinds, then greens, and finally on May 2 he expired (after throwing up black vomitus in very large quantities four times, and after presenting sweats chills and fainting spells) after his last attack of vomiting The autopsy showed that the wound had pierced the lungs and diaphragm

vigorous exercise Though trauma may be the immediate factor the underlying cause is a congenital predisposition

Herniation of the diaphragm may be of a progressive character so that the entire stomach may ultimately be contained within the hernial sac, drawing the colon along with it

patient turned in the first oblique position As the patient while in this position, swallows the barium suspension and the esophageal contour is delineated, a moderate amount of displacement of the cardiac end may be noted as a result of the pressure of the hernia By then overdistending the stomach with a large amount of barium and



Fig 541 Large hernia of the stomach through the diaphragm

ROENTGEN DIAGNOSIS

Herniation of the diaphragm may be recognized by (1) filling the stomach with barium and then (2) forcing the barium into the herniation by manipulation and change of position Such herniations of the stomach especially in their earliest manifestations may also be demonstrated by special study of the esophagus

Considerable information can occasionally be obtained regarding the presence of a herniation of the diaphragm on direct fluoroscopic observation An air bubble may be noted just above the diaphragm with the

manually forcing it upward the entire contour of the herniated region can be clearly brought to view Although examination in the erect position may be sufficient when the diaphragmatic hernia is large it may not infrequently escape detection unless examination is also conducted in the prone and occasionally in the Trendelenburg position This is particularly true when the herniation is small In the elderly small protrusions of the stomach through the esophageal hiatus are so common as to be considered almost a physiologic concomitant of old age

is without an enclosing hernial sac The most common sites in the probable order of frequency of occurrence are (1) through the hiatus pleuroperitonalis (foramen of Bochdalek) (2) through the dome of the diaphragm (3) through the esophageal hiatus (4) through the foramen of Morgagni and (5) through the gap left by partial absence of the hemidiaphragm

2 If acquired after birth, the sites of occurrence are (1) through the esophagus hiatus which type has an enclosing hernial sac (2) through the region of fusion of the anlage of the diaphragm and (3) at sites named under congenital types

through the esophageal opening there is a sac, but when through the leaf of the diaphragm there is usually no sac

2 In direct injury to the diaphragm, the hernia may occur at any point and is usually the result of penetrating wounds, such as those inflicted by a gunshot or a knife

3 Rupture of the diaphragm may be the result of inflammatory necrosis which in turn has been caused by a subdiaphragmatic abscess or rupture may follow necrosis from drainage tubes introduced into empyema cavities In these cases the opening in the diaphragm is usually posterior and there is no hernial sac



Fig 540 (A *Left*) Right oblique position Hernia of the stomach Note the displacement of the distal end of the esophagus (B *Right*) Same patient as is shown in (A) Position of the stomach after reduction of the hernia

Traumatic diaphragmatic hernia may be caused by direct or indirect injury or by inflammatory necrosis of the diaphragm

1 In indirect injury to the diaphragm, the hernia may occur at any point including points of embryologic fusion but the most common region is the dome and posterior half of the left hemidiaphragm it may however, also occur in the right hemidiaphragm It usually is the result of a severe crushing injury and the hernia may or may not have a hernial sac When the hernia is

Harrington also analyzed his findings in the examination of the esophageal hiatus in 1 000 cases during operation for other abdominal conditions and found that, in 8 per cent of the cases, 2 fingers could be inserted in the periesophageal space while, in 2 per cent 3 fingers could be introduced It is especially in these latter cases that hernial protrusions may develop, particularly in the presence of increased intra abdominal pressure due to injury, obesity pregnancy or as a result of vomiting or

at one time almost the entire stomach may be present within the chest and re examination at a later time may show that a good part of the stomach has returned to the abdominal cavity The heart may be displaced to the right and symptoms may be exaggerated by the increase in the degree of herniation and the consequent encroachment upon the pulmonary and cardiac areas With a descent of the stomach into the abdominal cavity, the displacement of the heart will become less marked and the symptoms may diminish or disappear completely into the chest The diagnosis is confirmed by examination through the administration of a barium enema which will then outline the entire colon and show the herniated loop above the diaphragm When loops of small intestine are suspected of partaking in the herniation examination by the ingested meal method may be essential

In the case of the colon, also there may be a sliding type of herniation, so that the colon may at times be noted above the diaphragm and at other times it may be entirely in the abdominal cavity In other

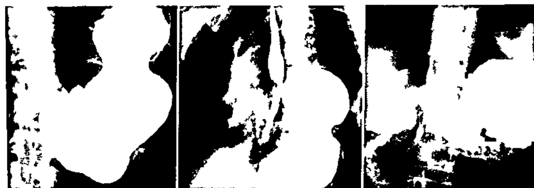


FIG 543 (A *Left*) Examination in the prone position at an angle of 90° Herniation through the dome of the diaphragm Note the inverted position of the stomach (B *Center*) Same patient as is shown in (A) Examination in the second oblique position Note the relation of the esophagus to the herniated portion of the stomach (C *Right*) Same patient as is shown in (A) and (B) Appearance after reduction of the hernia

pletely In some cases of even large hernias particularly of the congenital variety there may be a complete absence of any symptoms and the condition may be discovered only accidentally on routine roentgen examination

In addition to the herniation of the stomach itself through an opening of the diaphragm loops of bowel may also be present particularly the large intestine The first intimation that such a condition is present may occur in a roentgen examination of the chest Gas distended loops may then be noted above the diaphragm possessing the characteristic haustral markings in the case of the colon or the valvulae conniventes when loops of small intestine are crowded cases the herniation may be fixed permanently above the diaphragm The loops may appear as if partially incarcerated so that after evacuation of the barium distal to the hernia they may remain well distended for a long period of time

In rare cases herniation may actually involve the right diaphragm The stomach and the small and large intestine may partake in such herniation Well-controlled cases showing roentgen evidence of herniation through the right diaphragm have been reported by Breckoff [14] Kerr and Steinberg [1] Forty [16] Thomas[17] and Jenkinson and Roberts [18]

A case of bilateral diaphragmatic hernia was reported by May [19]

The herniated portion of the stomach will appear above the diaphragm, its exact location depending upon the particular region of the diaphragm which was involved

It is, of course, important to determine the exact relationship of the diaphragm and to demonstrate that the suspected hernia is above it in order to be certain that we



Fig 542 Note that the herniated portion of the stomach in this case is to the right of the esophagus

are not dealing with an eventration When the herniation occurs through the esophageal hiatus it becomes important to differentiate this condition from partial thoracic stomach and from ampullary dilatation of the cardiac end of the esophagus The important feature which distinguishes true herniation of the stomach is that the esophagus is of normal length, whereas in partial thoracic stomach, as previously described, the cause is a congenitally short esophagus A possible point of confusion is that the herniation of the stomach through the esophageal hiatus may carry the esophagus upward with it, making it appear shorter, although this is actually due to the fact that the cardiac portion of the esophagus has become tortuous and kinked

In distinguishing an actual herniation from an ampullary dilatation of the cardiac end of the esophagus, there are two points of importance One feature is the presence of mucosal folds in a gastric hernia characteristic of the rugal folds of the stomach The other point is that, if the ampulla is due to a dilatation of the distal end of the esophagus, it may be only transient and not present in all the films during a single examination or on repeated observations A fluoroscopic study of the behavior of the esophagus will also aid in such differentiation The mucosal relief of the dilated area may show longitudinal folds characteristic of the esophagus and continuous with the folds throughout the rest of the esophagus itself

At times, the herniated portion of the stomach, although entering the chest cavity by way of the esophageal opening, may swing over in such manner that it actually appears to be present in the right chest In such cases, the original examination of the chest may show a rounded shadow with a fluid level capped by gas in the region of the right lung, which may lead to an erroneous suspicion that a lung abscess is present Only by carefully following the course of the esophagus as it enters the stomach and then tracing the entire course of the barium will it be possible to determine the exact nature of this condition The prone and the Trendelenburg positions will be extremely helpful Not only will such examination serve to prove that the shadow in the right chest is due to a gastric hernia but it will also demonstrate that the hernia, although present on the right side must be classified as essentially of the paraesophageal type

A herniation may also roentgenologically show evidence of a sliding character Thus,

it admitted one finger through the opening Roentgen examination (Fig 537A) showed the herniated portion of the stomach through the diaphragm distended with barium Figure 537B showed the mucosal structure of this region with characteristic gastric rugae through the esophageal opening

The following case controlled by autopsy presented some interesting features

R M female aged 50 The patient complained of epigastric pain during the preceding 6 months which was relieved by food and bicarbonate of soda During the preceding 3 months she vomited blood on a number of occasions as well as on the night before her admission to the hospital She drank one pint of whiskey a day There was a loss of 30 pounds in the preceding year While in the hospital she had many transfusions but continued to hemorrhage and died

The autopsy report was On opening the peritoneal cavity there was a paraesophageal diaphrgamatic hernia 3 cm in diameter and 3 cm in depth A small portion of the stomach moved freely through the herniation Examination of the esophagus revealed moderate sized varices in the distal two thirds These varices communicated with varicose submucosal veins of the adjacent stomach At one point blood could be expressed from a varix into the lumen of the gastro intestinal tract in the region of the esophagogastric junction Blood clots were found throughout the alimentary tract No ulcerations of the stomach or remaining gastro intestinal tract were found

The final diagnosis was (1) portal cirrhosis of the liver (2) esophageal and gastric varices with rupture of esophageal varix with massive gastro intestinal hemorrhage (3) paraesophageal hernia

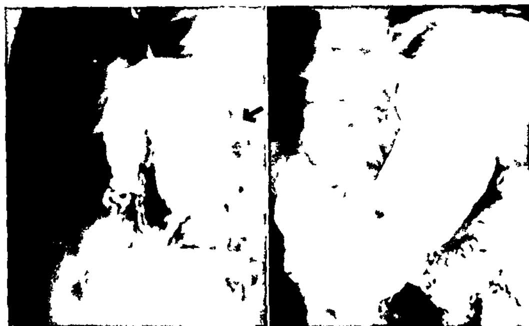


Fig 545 (A *Left*) Traumatic herniation of the entire stomach into the left chest Examination in the second oblique position Note the tube passing through the esophagus and entering the inverted pars cardiaca (B *Right*) Same patient as is shown in (A) Appearance after reduction of the hernia

Roentgen examination (Fig 5‹8) revealed evidence of a paraesophageal hernia The extreme distal end of the esophagus was obscured by the herniated portion of the stomach No evidence of the esophageal varices found at autopsy was noted at this time In this case the bleeding which might have been attributed to the hernia was actually due to esophageal varices

The importance of examination of the

ABSENCE OF THE DIAPHRAGM

The first definite case of absence of the left diaphragm proved by operation was reported by Harris and Clayton Greene[20]

The presence of intestinal loops within the chest may occur as a result of the absence of a leaf of the diaphragm In such cases roentgen examination in the lateral position may show an absence of any evidence of a shadow produced by the diaphragm Such recognition may be difficult however if the diaphragm has become extremely attenuated through atrophy as in cases of eventration of long standing Artificial pneumoperitoneum may be of considerable diagnostic help in such cases by showing an uninterrupted continuity of the air within the abdomen with that in the chest on the affected side

Pneumothorax may also be helpful in the absence of an entire leaf of the diaphragm, as well as in a diaphragmatic hernia by demonstrating the escape of air from the chest into the abdominal cavity

An extremely rare type of herniation is that of intestine into the pericardial sac, so that the coils are in direct contact with the heart[21] The diagnosis may be made by the recognition of intestinal coils within the confines of a distended pericardium

Illustrative Cases A hernia of the stomach through the esophageal hiatus (paraesophageal hernia) is illustrated by the following case



Fig 544 Herniation of the stomach Note the large diverticulum in the pars cardiaca

M E, aged 50 The patient gave a 3 months' history of epigastric discomfort, dull, intermittent in character made worse after a heavy meal At times the pain radiated to the back There was no weight loss

Physical examination of the abdomen was essentially negative except for the scar of a former appendectomy

The operative findings were as follows There was a gastric hernial protrusion through the esophageal hiatus in the diaphragm The size of the hernia was about the circumference of a half dollar The stomach wall was loosely adherent to the esophagus and to the diaphragm After displacing the herniated portion of the stomach into the abdominal cavity the hiatus was closed so that

right oblique position the herniated portion of the stomach is usually to the left of the esophagus it may rarely be present to the right (Fig 542)

Herniation of the stomach may not infrequently occur through other openings in the diaphragm than the esophageal hiatus as illustrated by the next case

B C male aged 38 The patient had originally complained of vomiting of 2 months duration This occured immediately or within 1 to 2 hours after meals He had been operated elsewhere for a hernia of the stomach and he was well for about 10 months when he had a recurrence of vomiting both solids and liquids The food seemed to stick in the epigastrium and he was relieved by vomiting There was no blood in the vomitus or stools He stated that he lost 100 pounds in several months

At operation by thoracic approach a large ring was found in the diaphragm measuring 3 cm in diameter to the left of the esophagus and through this ring was a herniated stomach which was incorporated in a fibrous sac The stomach was replaced into the abdominal cavity and the opening into the diaphragm closed One year later there was a recurrence of the herniation At this time fully 90 per cent of the stomach was found herniated into the left chest The ring of the diaphragm measured 2½ inches in diameter and the stomach was adherent to the ring by fibrous adhesions There was a well developed sac above the diaphragm

The operation was carried out through a transabdominal approach The incarcerated stomach was freed from the sac lying in the chest and returned to its normal site in the abdominal cavity



Fig 546 B Same patient as is shown in Figure 546 A Appearance after reduction of the hernia

Roentgen examination (Fig 543A and B) showed the esophagus to be of normal length The stomach was upside down and herniated into the left chest through an opening in the diaphragm to the left and independent of the esophageal hiatus Figure 543C shows the appearance after reduction of the hernia

The following is another example of herniation of the stomach through an opening

patient in various oblique positions is illustrated by the following case

D C, aged 53 The patient gave a history over a period of many years of recurring attacks of pain in the epigastrium radiating across the abdomen and to both shoulders without relation to food Some of the attacks had been very severe, on a few occasions requiring morphine There was no vomiting or blood in the stool Physical examination was essentially negative except for an old appendectomy scar

At operation there was a defect admitting 3 fingers at the esophageal hiatus through which the proximal portion of the stomach had herniated The herniated portion of the stomach was readily replaced into the abdomen and the defect in the diaphragm repaired

Roentgen examination (Fig 539A) revealed a fairly large hernia through the diaphragm The esophagus showed considerable serration of the contour and in this view gave the impression of a short esophagus entering the dome of the herniated portion of the stomach Figure 539B however, taken in the second or left oblique position, showed what had actually occurred The gastric hernia had displaced the distal end of the esophagus, giving the very definite impression that if the stomach were pulled down below the diaphragm, the esophagus would straighten out and assume its normal position It was, therefore, not a case of a congenitally short esophagus but one of true paraesophageal hernia, a diagnosis that was confirmed at operation



Fig 546 A Traumatic herniation of practically the entire stomach into the left chest associated with volvulus

The importance of the oblique position is further shown in the surgically confirmed case illustrated by Figure 540A The entire length of the esophagus may be clearly noted with the distal portion displaced by the hernia Figure 540B shows the position of the stomach after reduction of the hernia

The herniated portion of the stomach may be quite large (Fig 541) Although in the





Fig 549 (*Left*) Congenital herniation of almost the entire stomach
Fig 550 (*Right*) Incarcerated herniation of the colon



Fig 551 (A *Left*) Congenital herniation through the right diaphragm Appearance of the chest showing the presence of encapsulated gas on the right side (B *Right*) Same patient as is shown in (A) Appearance after filling the stomach with barium Note that the stomach is on the right side and that the pars cardiaca is herniated through the right diaphragm

in the diaphragm to the left of the esophageal hiatus

R B female, aged 24 During the preceding 2 years this patient had had attacks of vomiting unassociated with bleeding or abdominal pain She had lost 31 pounds during this time Physical examination of the abdomen was negative

At operation there was an egg shaped opening in the left leaf of the diaphragm measuring 2½ inches in its transverse diameter and 1½ inches in its anteroposterior diameter lower surface of the left lobe of the lung could be seen through the sac There was no collapse It was impossible to remove the sac and the *peritoneal surfaces* were liberally painted with iodine in the hope of stimulating *peritoneal adhesions* within the sac The opening was obliterated by *mattress sutures* of heavy silk The stomach was sutured to the diaphragm near the edges of the closed ring

Roentgen examination (Fig 544) revealed the presence of a large herniation of the stomach through the midportion of the diaphragm





Fig 547 (*Left*) Traumatic herniation of the stomach with a classical upside down appearance

Fig 548 (*Right*) Herniation of the stomach through the left diaphragm passing toward the right side

The hernial opening was 1 inch to the left of the *esophageal opening The edges of the ring* were firm and fibrous The sac contained transverse colon and about two thirds of the *stomach* The colon was adherent The stomach was fixed by adhesions which were delicate and easily separated The spleen was adherent to the parietal peritoneum immediately lateral to the defect in the diaphragm and was not part of the hernial contents The colon was easily reduced by traction following which the stomach was brought down into the abdomen by traction With the stomach held downward and to the right the edges of the ring could be *visualized* The There was no evidence of paradoxical respiration

Examination of the cardiac portion of the stomach showed the presence of a diverticulum originating from the lesser curvature border The narrow neck of the stomach as it passed through the diaphragmatic opening could be readily seen

Particularly as the result of trauma, the entire stomach may be herniated into the chest and may assume an *upside down* appearance

J R male aged 45 This patient was

pleura was opened the entire stomach, transverse colon and a portion of the omentum were found in the left pleural cavity herniating through a large rent in the diaphragm 3½ inches in length The organs were replaced and the tear repaired

Roentgen examination (Fig 545A exposure made in the left oblique position) showed the entire stomach above the left diaphragm and inverted in position Note that the tube passing through the esophagus entered the inverted pars cardia Figure 545B showed the appearance after recovery from the surgical procedure Five years later a follow up showed that the patient was entirely well

Herniation of the entire stomach into the chest after a bullet wound is described in the next case

F G female aged 21 Four years before her admission to the hospital the patient suffered from a bullet wound in the left side of the chest She remained well until 16 months before admission when she developed severe coughing spells and at times coughed up considerable blood She noticed tarry stools on one occasion During the month before admission she had attacks of severe epigastric pain after eating somewhat relieved by vomiting She had lost 15 pounds in the preceding year

Operative findings were Through the upper portion of the left dome of the diaphragm in the region of the esophageal hiatus there was a hole which measured about 1½ inches in diameter Through this hole the entire stomach had herniated The stomach was markedly adherent to the inferior portion of the lung to the mediastinum and most of all to the diaphragm in the immediate region of the opening There were a great many adhesions in the lower left pleural cavity so that after the lung was removed from the pleural cavity there was no tendency toward the collapse of the lung The entire stomach was freed and pushed back into the peritoneal



Fig 552 Herniation of the stomach through the right diaphragm as visualized in the right lateral position

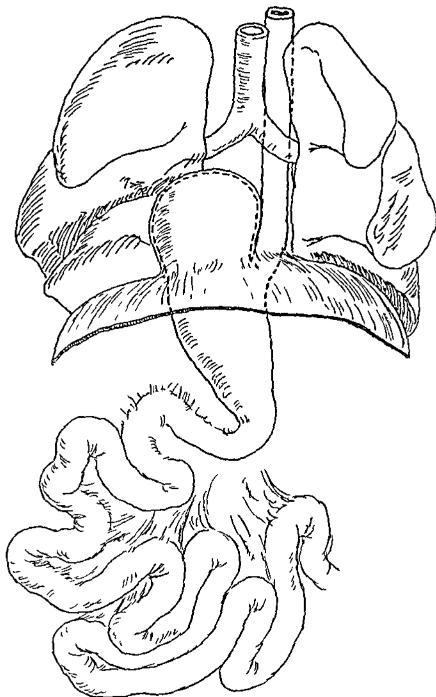


FIG 551 C Same patient as is shown in Figure 551 A and B Appearance of the right diaphragm and stomach as found at autopsy

severely injured in an automobile accident His ribs were fractured, as well as a number of transverse processes of the lumbar vertebrae He had recurrent severe left chest pain radiating to the left upper quadrant

Physical examination revealed an acutely ill white male The breath sounds over the left chest below the scapula were distant and this area was flat to percussion There was boardlike rigidity on the left side of the abdomen and marked generalized tenderness

At operation through the chest when the

In the following case the findings were controlled by autopsy

W S newborn This was a premature male child who appeared cyanotic from birth The cyanosis which increased in severity with crying and during feeding rapidly became worse and the infant died 12 days after birth

Autopsy revealed a congenital absence of the interventricular septum patent foramen ovale atresia of the pulmonary arteries and patent ductus arteriosus

The report on the gastro intestinal tract was There is marked distention of the entire bowel from the duodenum downward The visceral peritoneum is a little injected but smooth The stomach is rotated in such a manner that the greater curvature lies toward the right and the lesser curvature toward the left The pancreas maintains its anatomical relationship to the pylorus and duodenum but the main body of the gland lies behind the stomach slightly to the right and parallel to the long axis of the infant The upper one third of the stomach passes through a 2½ cm defect in the dome of the diaphragm just to the right of the midline and is loosely incarcerated and held by adhesions in a sac which protrudes above the diaphragm into the right thoracic cavity adjacent to the mediastinum for a distance of about 4 cm On opening the stomach the esophagus is found to open into the cardia below the diaphragm and is therefore apparently not shorter than normal The hernial sac is made up of 2 layers the upper leaf of diaphragmatic pleura and the lower leaf of the parietal peritoneum

Roentgen examination of the chest (Fig 551A) showed the presence of encapsulated gas in the right chest which was interpreted as being due to a herniated viscus Examination of the stomach showed it to be on the right side and the pars cardia herniated through the right diaphragm into the chest (Fig 551B)

Figure 551C shows the appearance of the hernia of the right diaphragm as found at autopsy

Figure 552 shows herniation of the stomach through the right diaphragm as visualized by examination in the right lateral position

The value of pneumoperitoneum in the demonstration of absence of a leaf of the diaphragm is illustrated in Figure 553 The air injected abdominally entered the right chest producing an artificial pneumothorax with compression of the lung Part of the colon was present in the right chest Note the delineation of the spleen within the abdomen by the cushion of air surrounding it

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cavity through the opening in the diaphragm and the rent in the diaphragm was repaired

Roentgen examination (Fig 546A) showed the herniation of practically the entire stomach through the left diaphragm with volvulus (upside down stomach) Figure 546B shows the appearance after the successful reduction of the hernia



FIG 553 *Absence of the right diaphragm* Appearance after artificial pneumoperitoneum The air introduced intraabdominally passed directly into the right chest

The upside down appearance of herniation of the stomach after severe trauma is also illustrated by Figure 547

The herniated distal portion of the stomach passing through the left diaphragm may swing over to the right side (Fig 548)

An example of herniation of almost the entire stomach into the thoracic cavity, apparently of congenital origin, is illustrated by the next case

F C, male aged 9 Examination revealed an undernourished child complaining of a productive cough He was referred to the hospital by an outside physician who, on routine examination found some abnormality of the left lung The general condition of the child was good He was noisy, active, bright and did not complain of dyspnea There was no evidence of cyanosis The findings on roentgen examination were obtained as the result of a routine study

Roentgen examination (Fig 549) revealed a herniation of practically the entire stomach In addition loops of small intestine were noted high up in the apical region of the lung It was noteworthy that in spite of the considerable encroachment upon the left chest, the child was symptom free the discovery of the abnormal condition having been made only as a result of routine roentgen examination

Herniation of the colon through the left diaphragm, operatively controlled, is shown in the next case

P P, aged 29 Six years before hospitalization this patient began having occasional sharp pains in the left upper quadrant of the abdomen These pains would last from 4 to 24 hours and would occur about once in 2 to 3 months At first the pain had no relation to food Six months before admission the cramplike pain in the left upper quadrant of the abdomen was associated with vomiting it radiated to the left shoulder and was aggravated by the ingestion of food Vomiting relieved the pain There was no blood in the vomitus On the night of admission to the hospital the patient had an attack of pain which was not relieved by vomiting

Physical examination of the abdomen was essentially negative except for slight tenderness in the left iliac region

Operation revealed the following 'The splenic flexure is noted passing through a small opening in the posterior part of the diaphragm and very close to the spinal column The mass cannot be reduced The transverse colon is very short The entire bowel is thickened and edematous'

Roentgen examination (Fig 550) revealed herniation of the splenic flexure through the left diaphragm into the left cavity of the chest There appeared to be considerable constriction of the distal transverse colon just before it communicated with the herniated portion of the colon The descending colon was also constricted where it emerged through the opening of the diaphragm

Herniation of the right diaphragm is rare

57

Eventration of the Diaphragm

ROENTGEN DIAGNOSIS

In eventration there is upward displacement of the diaphragm, the diaphragm however remains intact there being no openings through which any of the abdominal viscera can protrude into the chest The left leaf is more commonly elevated Jean Louis Petit is credited with being the first to have described a case of eventration of the diaphragm

The congenital nature of the eventration in some cases is shown (1) by its occurrence in the newborn (2) by the fact that in most cases it is on the left side resembling congenital herniation and (3) by the absence of any evidence of pressure on the lungs The lungs are small but normal in position The congenital nature of eventration of the diaphragm according to Verbrycke[1] is also suggested by the fact that the condition is often associated with other anomalies of a congenital nature and the absence of any other known causative agent It is possible however that injury to the phrenic nerve at birth may be an important factor in causing degeneration of the diaphragm and eventual eventration

The role of paralysis of the phrenic nerve in the causation of eventration was shown by Morison in two cases of this nature which came to autopsy In one case the phrenic nerve was involved in a cancer nodule Section of the nerve below this region showed complete degeneration Section of the diaphragm showed degeneration of muscle fibers In a second case there was involvement of the phrenic nerve close to the diaphragm Moreover eventration of the diaphragm can be produced artificially by severing the phrenic nerve

ROENTGEN DIAGNOSIS

In eventration fluoroscopic observation will show marked upward displacement of the diaphragm As a rule, the involvement is on the left side although in rare instances it is the right diaphragm that may be *affected*[3]

When the stomach is distended with barium it is noted that it remains entirely below the diaphragm there being no opening through which it can project into the thorax This is the important feature which differentiates eventration from actual herniation of the diaphragm

In some cases of eventration the respiratory excursions may show a reversal on the involved side the diaphragm moving upward with inspiration and downward with expiration This has been described as paradoxical respiration The occurrence of this phenomenon is to be explained by the fact that the thinned-out diaphragm is practically devoid of muscle It is essentially a fibrous structure covered by peritoneum below and pleura above and is therefore unable to contract During inspiration the right leaflet, normal in character descend There thus develops an increased intra abdominal pressure as a result of which there is an upward dislocation of the weakened left leaf of the diaphragm When the left diaphragm is high and a large gastric air bubble is present this may neutralize the increased intra abdominal pressure in such a manner that there may be little or no movement of the diaphragm In many cases however dependence upon paradoxical respiration offers no clue in differentia

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FIG 556 (A *Left*) Congenital eventration of the left diaphragm Examination in the erect position Note the air bubble of the stomach directly under the elevated left diaphragm (B *Center*) Same patient as is shown in (A) Examination in the prone position (C *Right*) Same patient as is shown in (A) and (B) Examination in the left lateral position showing the stomach beneath the left leaf of the diaphragm



FIG 557 (A *Left*) Eventration of the inner half of the right diaphragm shown by artificial pneumoperitoneum (B *Right*) Same patient as is shown in (A) Note the position of the stomach on the right side in relation to the eventrated diaphragm

In the next case of eventration of the left diaphragm a possible etiologic factor is roentgenologically demonstrable

H W male aged 36 For the past 3 years this patient had been complaining of distress immediately after meals lasting about ½ hour One week before his hospitalization this distress became much more pronounced He had lost 30 pounds in the preceding 2 months Physical examination revealed an old Pott s disease and tuberculosis of lymph nodes

Roentgen examination (Fig 555) revealed eventration of the left diaphragm In addition there were numerous irregularly calcified nodes throughout both sides of the neck The possibility arises that the eventration of the left diaphragm may have been acquired as a result of the involvement of the phrenic nerve on the left side by calcified nodes

tion between herniation and eventration of the diaphragm

Differential diagnosis may also be aided by artificial pneumoperitoneum In the case of eventration, no air would appear in the thoracic cavity, whereas in herniation air might escape into the chest In the case of a hernia, however which is closely bound to the diaphragm by adhesions, none of the air will be able to pass through the herniated opening Moreover the sac of the hernia, when present, will prevent the escape of air into the chest Examination by pneumoperitoneum, while occasionally helpful cannot of course be considered a routine method of differential diagnosis and its use should be limited only to those cases in which a decision as to surgical intervention may be imminent and when such differentiation may be altogether impossible in any other way

Both in herniation and eventration there is occasionally displacement of the heart and the mediastinum to the unaffected side the degree of displacement varying with the phase of respiration and the degree of distention of the displaced viscera

Illustrative Cases The following case is an example of eventration of the left diaphragm

R H aged 52 About 6 years previously this patient first noticed a squeezing pain about the heart while walking, this pain radiated up to the left shoulder and back The pain recurred about once a month until a month before hospitalization, when it became more frequent following physical exertion The patient had been dyspneic since the age of 18

Examination 3 years previously had shown herniation of the stomach through the esophageal hiatus and phrenectomy was performed The phrenic nerve was isolated over lying the scalenus anticus muscle

Roentgen examination (Fig 554) revealed high eventration of the left diaphragm with considerable displacement of the heart to the right There was no evidence of the herniation through the esophageal hiatus which had been noted 3 years previously It is possible that the eventration developed as the result of the phrenectomy particularly so since there was no evidence of eventration at the examination 3 years before





Fig 554 (*Left*) Eventration of the left diaphragm History of left sided phrenectomy Note the marked upward displacement of the left diaphragm

Fig 555 (*Right*) Eventration of left diaphragm Note many calcified nodes in the neck

THE GALLBLADDER AND BILE DUCTS

Congenital eventration of the left diaphragm is illustrated by the following case

L H, female, aged 5 weeks The infant was brought to the hospital for routine physical examination The child appeared poorly nourished and there was a loud systolic murmur over the precordium There was no cyanosis, but there was dyspnea of a moderate degree Small feedings were found necessary because of frequent vomiting The child showed a moderate weight gain under observation Roentgen examination (Fig 556A, erect, Fig 556B, prone) showed the stomach under the elevated left diaphragm Examination in the erect left lateral position (Fig 556C) showed the intact dome of the left diaphragm and the air bubble of the stomach directly under it

The value of pneumoperitoneum in delineating the diaphragm is illustrated by Figure 557A The left diaphragm is normal The right diaphragm shows an elevation at its inner border Figure 557B shows the appearance after filling the stomach with barium Most of the stomach is present, at the inner border of the diaphragm, on the right side There were no clinical symptoms referable to the condition

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THE GALLBLADDER AND BILE DUCTS

58

The Normal Biliary Tract

ANATOMY AND PHYSIOLOGY

ANATOMY AND PHYSIOLOGY

A knowledge of the anatomy and physiology of the biliary apparatus is of considerable importance to an understanding of many of its roentgen manifestations.

The intrahepatic ducts carry the bile from the liver to the right and left hepatic ducts. These then unite to form the hepatic duct which, passing downward for a distance of about 3 cm., is joined by the cystic duct, usually at an acute angle. In this manner is formed the common duct. The latter is about 7 cm. long and about 3 mm. in diameter and passes posteriorly to the superior portion of the duodenum and the head of the pancreas. Joining the pancreatic duct, the two open into the second portion of the duodenum at the papilla of Vater.

The gallbladder is pear shaped and lies in a fossa on the under surface of the right lobe of the liver. The lower part is the fundus, and it joins the cystic duct by a neck. The fundus is ordinarily directed downward and to the right and also anteriorly. The gallbladder is therefore in close relation to the liver at its upper surface, to the abdominal wall anteriorly, to the transverse colon inferiorly, and posteriorly to the transverse colon and the descending portion of the duodenum. Along its inner border it may lie in close relation to the duodenum and the pylorus. All these anatomic relationships have an intimate bearing upon various changes noted in the roentgenogram.

The cystic duct, passing downward and to the left, is characterized by the spiral valves of Heister. The gallbladder consists of a mucosa, a muscularis muscosae, a fibrous layer, a subserosa and a serosa.

The mucosa is thrown up into numerous folds, suggesting on anatomic grounds that its function is one of absorption. The epithelial cells secrete mucus. Small amounts of lipoid deposits may be present normally in the epithelial cells of the mucosa.

In the fibromuscular coat, well defined longitudinal, oblique and circular layers have been described. Halpert[1] considered the muscular layer of the gallbladder as similar to the muscularis mucosae of the intestine and believed that it functions primarily to produce the mucosal folds. A mucosa consisting of a single layer of columnar epithelium, a submucosa and a muscularis mucosae is present in the hepatic, cystic and common bile ducts. Muscle fibers are present at the duodenal termination of the common bile duct, and these are usually described as the sphincter of Oddi.

In describing the anatomy of the human gallbladder, Halpert stated that since the muscle bundles in the muscular layer do not form a compact layer throughout, the mucosa may protrude in some cases toward the external layers. This is particularly apt to occur where the vessels penetrate the muscularis to reach or leave the mucosa. These outpouchings of the mucosa are described as Rokitansky-Aschoff sinuses. These sinuses are very rare in the healthy human gallbladder and, when present, are shallow.

The gallbladder has an average capacity of about 40 cc. Its position varies with the general body architecture. In the hypersthenic it is usually globular and high in the right upper quadrant. In the asthenic

individual, the gallbladder is usually pear shaped elongated and may be present below the iliac crest

According to Boyden, Francis Glisson is credited with being the first to describe a sphincteric mechanism at the orifice of the common bile duct (1654) He stated that regurgitation of intestinal contents into the opening of the duct is prevented "by ring like fibers which occupy not only the opening of the duct itself but also the whole oblique tract" For the purpose of regulating the flow of bile into the intestine, he believed that another sphincter was responsible, located at the neck of the gallbladder The ampulla of Vater was first recognized by Abraham Vater in 1720 Gage[3] in 1879 described the muscular arrangement at the termination of the bile and pancreatic ducts This was followed by the classical contribution of Oddi,[4] who as a result of careful anatomic investigation demonstrated the existence of a ring of muscular tissue surrounding that portion of the common duct which traverses the intestinal wall He thereby excluded the possibility of its being simply a ring of circular intestinal muscle His conclusions were as follows

1 There exists a special muscular arrangement with a sphincter at the opening of the common duct into the intestine

2 This sphincter is formed of smooth muscle fibers and is largely independent of the muscular layers of the intestine

3 The probable function of this sphincteric arrangement is to render intermittent and to control the flow of bile into the intestine

4 At the opening of the duct of Wirsung there likewise exists a muscular arrangement with a sphincter

5 This sphincter arrangement accounts for several affections whose cause has not yet been determined clinically

Doyon[5] studied the muscular and nervous factors involved in the activity of the biliary apparatus He quoted Rudolphi and Muller as having observed rhythmic and peristaltic movements of the biliary ducts in birds Doyon confirmed these observations in pigeons and also in various mammals such as the dog He considered the phenomena of rhythmic and peristaltic movements as being independent of the central nervous system, since he was able to demonstrate these activities in the isolated organ He noted peristaltic activity in the gallbladder as the result of stimulation He found that the contractions of the gallbladder were similar to those of smooth muscle in general with a long latent period and progressive contraction of long duration In some cases, the contraction appeared to travel from one point to another in the manner of a peristaltic wave Similar observations were noted in the gallbladder of the rabbit and cat He also noted in experiments on dogs and cats contraction of the common bile duct under the influence of electrical stimulation of its walls He showed that stimulation of the end of the splanchnic nerves caused contraction of the gallbladder and inhibition of the sphincter of Oddi Stimulation of the central end of the vagus also caused contraction of the gallbladder and inhibition of the sphincter

Meltzer[6] similarly maintained that an antagonism existed between the muscle fibers of the gallbladder and those of the papilla During storage of bile there is contraction of the muscle fibers of the papilla and inhibition of the muscle fibers of the gallbladder When bile is discharged, the gallbladder contracts and the sphincter of Oddi relaxes

This reciprocal relationship received further support from the experimental work of Potter and Mann[7] and of Mc Master and Elman [8]

Disturbances in the law of contrary innervation may apparently lead to serious disorders of a functional nature such as colic, icterus, and even the formation of biliary calculi [9, 10, 11] This functional derangement in the reciprocal relationship of the gallbladder and sphincter has been described as biliary dyskinesia

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59

Gallstones, Limy Bile, Emphysema of the Gallbladder

TYPES AND LOCATION OF STONES

Biliary calculi may be classified as follows

1 Pure cholesterol stones These are formed of a mass of cholesterol crystals, are not stratified and will not show in the scout film prior to the administration of the dye for visualization of the gallbladder

2 Stones which are 75 to 90 per cent cholesterol, which are stratified and also contain a small amount of calcium Depending on the amount of calcium, some of these stones may cast a shadow in the roentgenogram

3 The most common type of stone, consisting of cholesterol and bile pigment These are as a rule, faceted and stratified

4 Bilirubin calcium stones, usually small in size and containing almost entirely bilirubin calcium and only rarely cholesterol

Rare forms are cholesterol stones forming about a nucleus of bilirubin calcium and stones consisting of calcium carbonate

When single, a calculus is usually of rounded contour and rough surface When present in the duct, however its form may represent a cast of the lumen When multiple, stones because of pressure against one another, become polyhedral with smooth, faceted surfaces

Most frequently stones are found in the gallbladder itself Occasionally they are found in the cystic, common and hepatic ducts, and at times also in the intrahepatic ducts

The size of the stone is of importance, since small ones carried along by the flow of bile from the gallbladder may enter the cystic duct and, in rarer instances, by way of the common duct enter the duodenum Larger stones obviously will not be as readily expelled through the papilla of Vater

When the stone becomes obstructed, it may ulcerate through the gallbladder or biliary passages, with the formation of a fistula leading into the duodenum and rarely into the colon and stomach It may then pass through the rectum, or if unable to get through the small bowel, may be one of the causes of intestinal obstruction

HISTORICAL DEVELOPMENT IN ROENTGEN DIAGNOSIS

Among the earliest attempts to radiograph biliary calculi was that of Gilbert, Fournier and Oudin[1] They presented before the Biological Society of Paris in 1897 the results of their efforts to radiograph calculi of varying chemical composition They found that while roentgenography of a cholesterol calculus showed hardly any evidence on the plate, those calculi which were rich in pigment were more readily demonstrable They placed 2 calculi of rather large size behind a leg which they radiographed by an exposure of 15 minutes, without being able to demonstrate the calculi on the plate They had a similar

experience in attempting to radiograph another calculus rich in pigment and placed in front of the thorax They concluded that it would be difficult to recognize the presence of calculi within the abdomen by the roentgen method

Morris Senior Surgeon to the Middlesex Hospital, writing in 1896 found it impossible to radiograph renal or biliary calculi in vivo The interposition of the ribs the position of the kidney close to the vertebral column the depth of the cavity and the thickness of the overlying tissues are as yet obstacles which possibly in the near future will be overcome

Eder and Valenta[3] in 1896 radiographed gallstones after removal from the body though they failed to do so while they were still within the abdomen

An interesting account in the historical development of the roentgenology of biliary calculi is to be found in the demonstration by Neusser[4] He showed one picture of a gallstone which had been radiographed through a layer of liver 4 fingers thick In discussing this presentation the Vienna correspondent of the Munchener medizinische Wochenschrift stated that since it took 1 hour to obtain a roentgenogram of a hand it could hardly be calculated how long it would take to penetrate the body for visualization of a gallbladder provided that these rays would go through such thick layers of tissue at all He wrote as follows

Every method hence also this one leaves room for improvement and if today it seems to us that what we hear is music of the future all these experiments nevertheless are very much worth while

Prompted by the publications of Neusser Buxbaum[5] attempted to radiograph calculi in the living This he succeeded in doing apparently for the first time and demonstrated the plates obtained in one of his cases at a Congress in Moscow It is interesting to note that in 1 case he found it necessary to make a roentgen exposure of 12 minutes Included in his article is a reproduction of a roentgenogram showing a faint but fairly definite opaque shadow in the region of the gallbladder

With improvement in technic a number of pioneers began showing biliary calculi within the abdomen

ROENTGEN DIAGNOSIS

In a small percentage of cases gallstones may be noted in the preliminary film when there is a deposit of calcium in them The roentgen appearance of the calculi will, of course vary with their pathologic characteristics They may therefore appear rounded faceted or slightly irregular in outline A calculus may show a homogenous density or only a thin rim of calcification, or lines of stratification depending upon the amount of calcium and its distribution through the stone

There may be only a solitary calculus or myriads of calculi may completely fill the gallbladder and outline its contour The solitary calculus may be very large filling the gallbladder and at times may be indistinguishable from calcification of the wall of the gallbladder In some cases several calculi may be definitely noted in the roentgenogram No attempt should be made however to predict the exact number of calculi actually present within the gallbladder from the number noted in the roentgenogram Not uncommonly additional calculi are present in the gallbladder which were not observed either because they were obscured by a more anteriorly placed larger calculus or because some of them did not contain sufficient calcium to cast a shadow This is one reason why it is practically impossible to determine whether any therapeutic measure has been effective in diminishing the number or density of any calculi in the gallbladder When re-examination apparently indicates that fewer calculi are present it may mean only that some of the calculi have been obscured by others

A study of the gallbladder area will sometimes show the marked variability in the relative position of the calculi A stone within the cystic duct however, may appear

fixed on repeated examinations This shifting change in the localization of calculi has a twofold significance (1) of clinical value is the fact that such changeability gallbladder, the relative position of which remains fixed This differentiating feature, of course, depends upon cholecystographic study, since the gallbladder tumor itself is



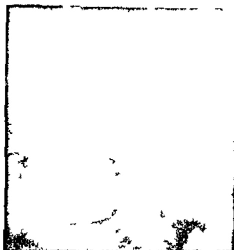


Fig 558 (*Left*) Solitary biliary calculus seen in the preliminary film
Fig 559 (*Right*) Multiple biliary calculi seen in the preliminary film



Fig 560 (*Left*) Multiple biliary calculi and a renal calculus in the same patient seen in the preliminary film



Fig 561 (*Right*) Gallbladder packed with calculi seen in the preliminary film

gives us an insight into the mechanism which leads to the eventual escape of a calculus into the cystic duct (*2*) the freedom of mobility of intrabiliary calculi is an aid in differentiation from a tumor of the recognizable only in this manner At times, however, calculi may be so fixed to the gallbladder wall as to show no change in position on repeated examination

On examining a patient for the presence

of biliary calculi the colon should be emptied by an enema the purpose being to eliminate fecal material because it may obscure the field of vision and because small fecaliths may be the cause of shadows in the right upper quadrant As in roentgen examination of the gallbladder in general immobility of the patient with complete cessation of respiration the use of intensifying screens and a Bucky diaphragm are essential The patient is in the prone position although in rare instances a slight 10 by 12 film may be equally satisfactory if the patient is small The important point is that one must not assume that biliary calculi are to be found only in the region of the costal margin since they may be present almost anywhere on the right side of the abdomen, occasionally even well below the iliac crest Moreover since we are interested in finding not only biliary calculi but also intrahepatic calculi, it is of value to include also the region of the liver In fact a scout film (size 14 by 17)



Fig 562 (A *Left*) Biliary calculi within the gallbladder and a calculus in the cystic duct seen in the preliminary film (B *Right*) Same patient as is shown in (A) Note the rearrangement of the calculi in the gall bladder

change in the angle of obliquity may bring a calculus into clearer relief when it lies close to the vertebra In some cases when the calculus is overlapped by a rib varying the position of the rays will cause it to appear in the intercostal space

The use of very small films for roentgenography of the gallbladder area is not recommended since calculi may show considerable variability in position A film large enough to extend from above the tenth rib to below the crest of the ileum should be employed The 11 by 14 film is used routinely in examinations of the gall bladder at Bellevue Hospital although the including the entire abdomen may be of considerable value in some cases as a preliminary procedure

Besides the presence of fecaliths differentiation of biliary calculi from renal calculi calcified lymph nodes and calcific deposits in the ribs may at times be necessary Differentiation from a fecalith depends not only on the fact that as a rule the latter bears no resemblance to the structural peculiarities of a calculus but also will be inconstant in location as the intestinal contents are shifted and will be completely eliminated by cleansing of the colon A renal calculus is often branched

and irregular and, as a rule, of increased density The shadows of urinary calculi may follow the anatomic course of renal structures, and in a well exposed film will be noted as being present within the soft tissues of the kidney itself

A gallstone shows to best advantage when the rays are directed postero anteriorly, a renal calculus shows best with the rays directed anteroposteriorly In rare instances however a renal calculus may stimulate a biliary calculus so closely as to make differentiation possible only by pyelography or cholecystography

A calcified lymph node is of irregular shape and should ordinarily offer no difficulty in differentiation because of its dissimilarity to the shadows of biliary calculi Calcifications in the course of a rib occur, as a rule, in the cartilaginous area and are frequently of bizarre shape and the continuity of the shadow may be traced with the rest of the rib When any suspicion exists regarding the significance of any opaque shadow which may be confused with a biliary calculus, we possess today a powerful weapon for accurate differential diagnosis in the cholecystogram

Illustrative Cases Figure 558 shows an example of a solitary calculus which was noted in the preliminary film Figure 559 shows an example of multiple calculi Figure 560 shows the presence of multiple biliary calculi and a renal calculus in the same patient

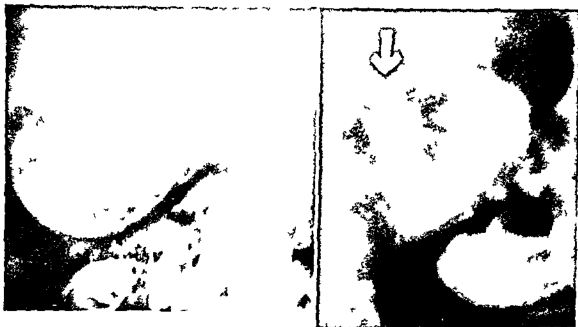


Fig 563 (*Left*) Calcification of the gallbladder
Fig 564 (*Right*) Pressure on the duodenal bulb produced by the gallbladder containing calculi

The following case is of a gallbladder completely filled with comparatively small calculi which outline its entire contour

D T female aged 21 This patient had her first attack of right upper quadrant pain 4 years previously following pregnancy She then had occasional attacks of right upper quadrant pain occurring about 1 to 6 months apart On the morning of her admission to the hospital she had a very severe attack of pain associated for the first time with vomiting The pain always radiated to the tip of the right scapula There was marked intolerance to fatty food but never any jaundice or

any clay-colored stools Physical examination disclosed tenderness in the right upper quadrant

Operation revealed a slightly thickened gallbladder containing numerous calculi Cholecystectomy was performed

The pathologic diagnosis was cholelithiasis with mild chronic cholecystitis

Roentgen examination (Fig 561) revealed a gallbladder completely filled with many small calculi There was a calculus in the cystic duct

Calcification of the gallbladder may be demonstrated in the preliminary film as is illustrated in Figure 563

The pressure on the duodenum produced by a gallbladder containing calculi is illustrated in Figure 564

LIMY BILE

An interesting though uncommon condition of the gallbladder is due to the pres-





Fig 565 (A *Left*) Appearance of the resected gallbladder Compare with (B) (B *Right*) Same patient as is shown in (A) Limy bile associated with numerous calculi in the gallbladder and a calculus in the cystic duct

The mobility of calculi within the gallbladder is illustrated in Figures 562A and B In a comparatively brief interval between examinations there was a definite realignment of the calculi within the gallbladder The calculus at the cystic duct was essentially identical in both films The presence of the calculus in the cystic duct would also help to explain the failure of visualization of the gallbladder after the administration of the dye The calculus blocking the cystic duct prevented the dye excreted by the liver from entering the gallbladder This case was confirmed at operation

ence of lime containing bile so that it becomes roentgenographically visible in the scout film prior to the administration of the dye The first observation of calcium containing bile was by Churchman[6] On opening a gallbladder at operation he found in addition to several calculi a grayish white glistening opaque mass of the consistency of tooth paste Chemical examination showed this substance to consist of soaps of the calcium salts

This lime containing bile was later described by Volkmann as lime milky bile He considered the terminology justifiable

because the bile in the particular gall bladders that he examined appeared as a *thin, white fluid In some cases, however,* the bile may be pasty and even solid More over, the color may vary, being at times yellowish or even dark brown Knuttson,[8] therefore, considered Volkmann's term "lime milky bile" inappropriate and de scribed the condition as "limy bile" This descriptive term can be used to refer to the This of course, will occur only when the limy bile is not puttylike but fluid in nature

Frequently a stone in the cystic duct can be noted in such cases This has been true in every case of limy bile that I have personally observed Calculi may be noted within the gallbladder, imbedded within the limy bile itself At times the roentgenogram may give the impression of a dye

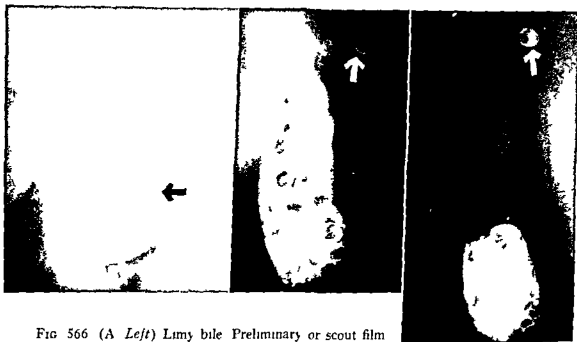


FIG 566 (A *Left*) Limy bile Preliminary or scout film of the gallbladder area Note the opaque shadow at the lower pole including a few vaguely outlined translucent areas within its confines (B *Center*) Same patient as is shown in (A) Roentgen examination of the resected specimen taken in the horizontal position (C, *Right*) Same patient as is shown in (A) and (B) Roentgen examination of the resected specimen, suspended In particular, compare this appearance with that of (A) Note also the stone in the cystic duct in (B) and (C)

condition regardless of the exact color or consistency of the bile *It refers only* to the abnormally limy character of the bile and its demonstrability as a dense calcium shadow in the roentgenogram Additional descriptions of limy bile were those of Phemister Rewbridge and Rudisill[9] and Kornblum and Hall[10]

The limy bile may at times show a change in appearance exhibiting a horizontal upper level *in the erect position* with an absence of such a level in the horizontal position[11]

filled gallbladder in those cases in which a preliminary scout *film has not been taken* prior to the administration of the dye However the fact that there is no change whatever in the appearance of the gallbladder after the fat meal should create the suspicion that the density of the gallbladder is due to the presence of limy bile Examination without the administration of the dye will clear up the diagnosis This is one reason among others why the taking of a scout film prior to the administration of

any dye should be part of the routine examination of the gallbladder

Illustrative Case The following is an example of the findings in a case of limy bile





FIG 567 (A *Left*) Limy bile Appearance in the preliminary film (B *Right*) Same patient as is shown in (A) Roentgen examination of the resected gallbladder

M D female aged 53 This patient gave an 8 year history of attacks of substernal pain radiating to the back never associated with vomiting chills or fever She had lost 7 pounds in the preceding year Cholecystectomy was performed Gross examination revealed a gallbladder of about normal size and with thin wall packed solid with faceted calculi cemented together by yellowish white chalky material A calculus was also present in the cystic duct (Fig 565A) Microscopic examination revealed extensive denudation of the lining epithelium Many of the glands were displaced within the wall of the gallbladder There was no fibrous tissue deposit and no active inflammatory exudate in the wall Chemical examination of the bile showed it to contain calcium carbonate volumes per cent 88 Practically all the stones were calcium stones

Roentgen examination (Fig 565B) without the administration of any dye showed the gallbladder to contain many calculi A moderate amount of dense material was present mainly at the lower pole of the gallbladder There was a rounded calculus apparently at the neck of the cystic duct The appearance of the gallbladder was identical after the administration of the dye and after the fat meal The preoperative diagnosis was cholelithiasis with limy bile and this was confirmed at operation as noted above

The diagnosis of limy bile in this case was made on the basis of the roentgen appearance of the preliminary film showing the presence of the dense calcific material at the lower pole of the gallbladder as if it had actually been filled with radiopaque concentrated dye It is also of some interest that a calculus was present in the cystic duct Neither the administration of the dye nor the fat meal produced any alteration in the appearance of the gallbladder

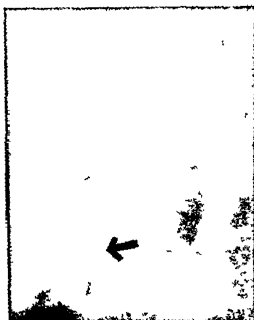


FIG 568 Limy bile Note the opaque shadow produced by the limy bile within the gallbladder in the preliminary or scout film of this region There are a few vaguely outlined translucent areas within the dense shadow due to the incorporation of several calculi

Some of the characteristics of limy bile are illustrated in the next case

M P , female, aged 39 During the preceding 2 weeks, the patient had had 3 attacks of severe nonradiating pain localized to the right upper quadrant and epigastrium This was accompanied by vomiting which was followed by relief Each attack lasted about 6 to 8 hours Physical examination was essentially negative except for tenderness in the right upper quadrant

A cholecystectomy was done The common duct was explored and several small stones and "mud were removed A T tube was inserted The gallbladder contained a considerable amount of mud and approximately twenty small stones The roentgenogram prior to cholecystography (Fig 566A) showed an opaque matrix in which calcific areas were imbedded

The preoperative diagnosis was limy bile with calculi There was no evidence of a stone in the cystic duct in the preoperative roentgenogram Because of the fact that every case of limy bile in our experience has been associated with a stone impacted in the cystic duct, it was predicted that such a calculus would be found at operation This may be noted in the roentgen reproductions of the resected specimen

Figure 566B shows the appearance of the intact gallbladder filled with ' limy mud and a stone in the cystic duct (gallbladder lying flat) Figure 566C shows the appearance of the gallbladder suspended Note that the limy menstruum and the calculi embedded within it have settled to the lower pole The calculus in the cystic duct is unchanged



Fig 569 (A *Left*) Limy bile Examination in the prone position (B *Right*) Same patient as is shown in (A) Examination in the erect position Note the change in the appearance of the limy bile due to the fluidity of the medium The upper arrow points to the stone in the cystic duct

In the next case the inspissated limy bile was impregnated with numerous tiny calculi

S D , aged 53 The patient gave a 15 year history of recurring attacks of pain in the epigastrium radiating to the right upper quadrant and at times associated with chills and slight fever but no vomiting Physical ex

amination was essentially negative except for tenderness in the right upper quadrant A cholecystectomy was done Examination of the content of the gallbladder showed inspissated bile in which numerous small calculi were impregnated

A preliminary film (Fig 567A) prior to cholecystographic study showed opaque substance in the location of the gallbladder with small vaguely outlined translucent areas in this region The preoperative diagnosis on the basis of this appearance was limy bile with numerous calculi imbedded in it Figure 567B shows the roentgen appearance of the resected gallbladder

The limy bile may be of rubbery consistency

N A male aged 40 The patient gave a 9 month history of attacks of very severe pain localized to the right upper quadrant These attacks awakened him from sleep about 5 A M and required a hypodermic of morphine for relief There was no vomiting chills fever or jaundice He lost 10 pounds Physical examination was essentially negative except for tenderness in the right upper quadrant

A cholecystectomy was done When the gallbladder was opened considerable limy bile was found to be present this was yellowish in color and of a somewhat rubbery consistency Within this limy bile were several small calculi A preliminary film without cholecystography (Fig 568) revealed an elongated somewhat pear shaped densely opaque shadow in the region of the gallbladder One of the films showed a small rounded opaque shadow in the region corresponding to the cystic duct When the gallbladder was opened there was no evidence of a calculus impacted in the cystic duct It is possible of course that during manipulation the stone may have been forced back into the gallbladder

Limy bile may show a change in contour depending on the position of the patient

R P female aged 72 The patient had several attacks of pain in the right upper quadrant one of which waking her from sleep required a hypodermic of morphine A cholecystectomy was done A stone was impacted at the mouth of the cystic duct Within the gallbladder itself the bile was puttylike and embedded within it were several concretions

Roentgen examination preliminary to the administration of the dye showed the following In the prone position (Fig 569A) there was a stone apparently at the mouth of the cystic duct The gallbladder itself contained opaque material and a number of irregularly outlined dense concretions In the erect position (Fig 569B) the opaque material within the gallbladder sank to the lower pole indicating considerable fluidity of its content

The diagnosis was limy bile in a fluid state

EMPHYSEMA OF THE GALLBLADDER

Infection by means of a gas producing organism may result in emphysema of the gallbladder a condition which in rare instances has been detected on roentgen examination Stolz[3] in 1901 described three cases examined at autopsy in which gas was encountered in the biliary tract He found two types of micro organisms the Frankel bacillus and bacilli of the coli group *Bacterium coli commune* and *Bacillus lactis aerogenes* There was no proof however that the gas infection was present before death since these were all post mortem findings

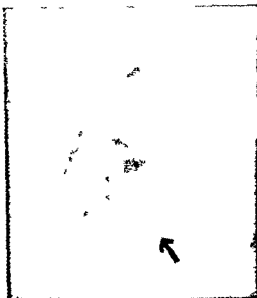


Fig 570 Emphysema of the gallbladder

Kirchmayr[13] in 1925 described the operative findings in a case of gas in the gall bladder The gallbladder was the size of a

fist and was adherent to the duodenum and hepatoduodenal ligament The wall was necrotic When the gallbladder was freed, *a malodorous gas escaped from it* The gallbladder was dilated and edematous, with multiple abscesses in the wall, evidence of an acute phlegmonous inflammation The patient recovered The Frankel Welch bacillus was *found on culture*

Bacillus welchii may be present normally in the gallbladder Andrews and Henry[14] showed that in one of six normal gallbladders *Bacillus welchii* was cultured from the wall In twelve normal gallbladders containing stones *Bacillus welchii* was obtained from the wall in two cases, and from the bile itself in one The organism was recovered from the bile in three out of four cases of obstruction of the common duct With the onset of acute infection of the gallbladder *Bacillus welchii* may become virulent and cause gas formation

The first roentgen evidence of gas in the gallbladder and in the pericholecystic region was described by Hegner[15] in 1931 *The amount of gas in the gallbladder* increased under observation after a period of 2 days At operation the gallbladder was under extreme tension because of gas present within it At autopsy the gallbladder *was found to contain* twelve calculi in addition to a calculus which apparently had extruded through a perforation near its neck A pure culture of an atypical *Bacillus welchii* was recovered from the field of operation Similar roentgenologic findings were described by Del Campo and Otero[16]

A remarkable study of the roentgenologic *findings of gas in the gallbladder* was reported by McCorkle and Fong[17] Included in their article are three cases in which the gas in the gallbladder had resulted from infection In the first case, in which a cholecystostomy was done, the patient died Culture showed *Bacillus welchii* In the second and third cases roentgenograms were taken at intervals and the development and increase of the gas in the gallbladder was traced, as well as the regression, until recovery under conservative management During the active phase the spread of the gas could be traced from the gallbladder into the surrounding tissues The authors also demonstrated in their roentgenograms the presence of a fluid level surmounted by gas within the gallbladder when the examination was made in the erect position

All three of Stevenson's cases[18] showed gas in the lumen of the gallbladder, in the wall and in the pericholecystic tissues In *two of the cases operation disclosed a gan*grenous gallbladder Remarkably enough the third case with roentgen findings of an essentially identical character underwent regression with conservative management

Figure 570 is an example of emphysema of the gallbladder (courtesy of Dr C F Hegner)

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60

Cholecystographic Diagnosis of Gallbladder Disease

DIRECT VS INDIRECT SIGNS OF GALLBLADDER DISEASE

The indirect signs of gallbladder disease deal mainly with evidence of fixation and displacement of the stomach and duodenum and of pressure of a distended gallbladder upon the duodenum, pyloric antrum and occasionally the colon

The indirect evidence of gallbladder disease, apparently due to fixation, displacement and pressure may be mimicked by so many other factors that conclusions *based on such evidence are as a rule* presumptive and often lacking in a sufficient degree of certainty Moreover such indirect evidence may be absent in the presence of actual serious disease of the gallbladder

Evidence of pressure upon the duodenum or pyloric antrum may occur under normal conditions and therefore cannot be accepted as evidence of gallbladder disease This is true for two reasons (1) a normal gallbladder may lie in such close relation to the pyloroduodenal region as to be capable of producing a pressure defect and (2) such pressure may be exerted by structures other than the gallbladder, such as the hepatic flexure of the colon

Particularly since the advent of cholecystography less emphasis is placed on indirect evidence of disease of the gallbladder and more on the direct signs of abnormalities of structure and function

HISTORICAL DEVELOPMENT OF CHOLECYSTOGRAPHY

The historical development of cholecystography is extremely interesting

Abel and Rowntree,[1] in their experimental observations on the phthaleins wrote as follows In the above experiment the interesting fact was brought to light *that the tetrachlor derivative is excreted* in the conjugated form in the bile before it appears in the 'free state They also recorded the fact that phenolphthalein and *its halogen substitution products, phenol* tetrachlorphthalein and tetrabromphenol tetrachlorphthalein do not differ markedly in pharmacologic behavior that their toxicity is very low and that large quantities may be injected repeatedly into a vein of the dog without causing any discoverable pathological lesions When the tetrachlor derivative is given subcutaneously, it escapes from the body in the bile only, the amount that passes into the intestine in other secretions being so minute that it can barely be detected

One of the great steps in the development of abdominal roentgenology was accomplished when Graham and Cole[2] in 1924 published their preliminary report on a new method of visualization of the gallbladder based on the fundamental work of Abel and Rowntree In their short but classic paper they wrote as follows

The revolutionary effect on the diagnosis of gastrointestinal conditions which was made by the use of the opaque meal has given rise repeatedly to the idea that if by some means an opaque substance could be safely introduced into the gallbladder, so that its contour could be seen with the roentgen ray the diagnosis of many obscure and doubtful cases of cholecystitis might be made easy and accurate To fulfill the necessary practical requirements the opaque substance must be something that is excreted into the bile after being given by mouth and furthermore it must be devoid of toxic effects when used in the concentration necessary for the shadow of the gallbladd r to be seen The extensive use of various dyes in a earch for a test of liver function has revealed the fact that certain ones are excreted almost entirely into the bile as for example tetrachlorphenolphthalein and ro e bengal Preliminary tests of intravenous injections of the sodium salt of tetrachlorphenolphthalein into experimental animals failed to reveal satisfactory roentgen ray shadows of the gallbladder Because of the similarity of chemical structure it was thought that tetra iodophenolphthalein would also be excreted into the bile and might cast a satisfactory shadow because of its iodine content Roentgen ray shadows of the gallbladder were revealed but the substance appeared to be too toxic to permit its extensive use in man Accordingly it was felt that tetrabromphenolphthalein might be a suitable compromise between the toxic iodine compound and the less opaque chlorine compound Th experimental results with this substance were satisfactory so far as toxicity was concerned but in some instances the shadows of the gall bladder were not so definite as was to be desired

It was therefore decided to try the calcium salt instead of the sodium salt of tetrabromphenolphthalein This substance when injected intravenously has given definite and cleanly cut shadows of the gallbladder both in experimental animals and in human subjects It is possible that some other substance will prove to have advantages over calcium tetrabromphenolphthalein

Cholecystograms accompany the article They also stated that they were able to obtain shadows in all cases of patients whose gallbladders were presumably normal They found difficulty however in obtaining a shadow in the case of a pathologic gallbladder They predicted that this very fact would prove to be an aid in the diagnosis of gallbladder disease

They soon found that the calcium had no particular advantage and that it was more practical to give the sodium salt [3] Later experience showed that although the first samples of sodium tetraiodophenolphthalein were too toxic for practical u e the purified product was no more toxic than equal doses of tetrabromphenolphthalein A smaller dose of the iodine containing compound was sufficient for cholecystography than of the bromine containing compound This is due to the greater atomic weight of iodine as well as to the fact that the iodine forms a greater proportion by weight of its compound than does bromine

Cholecystography as a diagnostic procedure seized the imagination of the profession and Moore [4] in February 1925 Wilkie and Illingworth of England in December 1925 and McCoy and Graham [6] in 1926 were able to show the remarkable accuracy of cholecystography in the diagnosis of gallbladder di ease

Oral Cholecystography A great advance in the popularity of cholecystography and its widespread application resulted from the development of the oral method of administering the dye

Menees and Robinson [7] in April 1925 showed that the oral administration of sodium tetrabromphenolphthalein was a practical procedure and that it could be given with safety in amounts necessary for visualization of the gallbladder Their original recommendation was to administer dye in capsules so as to minimize any irritating effect on the stomach Bicarbonate of soda was either placed in the capsules or given separately Films were taken at approximately from 12 to 14 hours and 18 and 24 hours later Soon the oral method of cholecystography became universally recognized as a practical procedure [8, 9, 10]

The oral method is superior to the intravenous method because of the simplicity of administration and the absence of danger of phlebitis or of extravasation of fluid at

the site of injection Reactions such as nausea, vomiting, diarrhea, headache and abdominal cramps, which occur after the administration by either method, are as a rule inconsequential, and even when marked, rarely last more than a few hours The disadvantage of the oral method is that *the degree of absorption of the dye cannot be as perfectly controlled* as with the intravenous method When the dye is *administered intravenously, the gallbladder* begins to be visualized in 4 hours There is a gradual increase in the density, with a shadow exhibiting *maximum concentration* at 12 hours When administered by mouth, a considerably longer period of time elapses before the gallbladder can be visualized As a rule, a cholecystogram can be obtained in 12 hours, although, frequently excellent concentration of the dye cannot be obtained until 18 hours after its ingestion

A further advance in cholecystography was made with the development of the method of repeated or intensive dosage of the dye as outlined by Sandstrom[11] and later by Stewart and Illick[12] The value of the administration of repeated doses of the dye was attributable to the fact that, occasionally following the oral administration of the dye, if only one dose had been administered no shadow of the gallbladder was obtainable, although at operation the *gallbladder was found to be normal* The cumulative effect of the administration of repeated doses of the dye at specific intervals overcame this serious discrepancy in the procedure and led to excellent visualization of the gallbladder, which could not be seen after the administration of only a single dose

The reason for the value of the intensive dose method of examination of the gallbladder is as follows The dye after its elimination from the liver and bile is reabsorbed by way of the intestine into the blood stream and again *reaches the liver* where the cycle of excretion is repeated By the additional administration of the dye *during this cycle*, we can obtain the cumulative effect of this together with the dye still circulating in the blood stream Not only is the method of great value in that a normal cholecystogram is obtainable when a single dose was entirely ineffective, but it obviously also aids in those cases in which the original shadow is of poor quality

Antonucci[13] described a technic of direct intravenous administration of glucose solution 10 minutes prior to the injection of the dye, a procedure which he believed caused a more rapid appearance of the *cholecystogram This view, however* was not corroborated by the work of Jung and Moore[14]

Another procedure recommended for the more rapid visualization of the cholecystogram and the determination of its distensibility was that of the intravenous administration of decholin[15,16,17] However, the intensification of the gallbladder shadow by the cumulative dose method and its response to the fat meal is in itself a sufficiently acceptable procedure and the intravenous administration of decholin unnecessarily complicates the examination

The ability to obtain a normal cholecystogram after the administration of the dye depends on the concentration of the dye after it reaches the gallbladder The mechanism involved was demonstrated by Rous *and McMaster*[18] *They showed experimentally* that bile coming from different portions of the liver of the dog has about the same amount of pigment Using pigment strength as a criterion they showed that the gallbladder exerts a rapid and very marked concentrating function Thus in one experiment a gallbladder which was emptied at the beginning and then permitted to fill with bile from the liver, concentrated 40 8 cc of bile to 4 6 cc *in the* course of 22½ hours This resulted because of the withdrawal of fluid through the wall *of the gallbladder* The bile ducts however do not withdraw fluid from the bile but on the contrary, tend to dilute it with a thin colorless secretion

Copher[19] demonstrated the excretion of bromine and iodine in the bile following the intravenous administration of sodium tetra bromphenolphthalein or tetra iodophenol phthalein the substance finally entering the gallbladder by way of the cystic duct and eventually becoming sufficiently concen trated to cast a shadow in the roentgenogram The maximum degree of concentra tion was attained in from 16 to 24 hours after injection He showed that the con centration of bromine in the bile before entering the gallbladder was 0 2 per cent Compared with this the average concentra tion of bromine in the gallbladder at the time when it cast a shadow in the roent genogram was 0 6 per cent This concen trating power of the gallbladder was verified by Ivy [0] According to him the normal gallbladder concentrates from 4 to 10 times the hepatic bile which it receives primarily as the result of the absorption of water

Boyden[21] fed cats on a diet of egg yolk and heavy cream and noted that the gall bladder exhibited the following functional periodicity a period of slow emptying a collapsed or resting period and a period of slow filling and bile concentration When pure protein in the form of egg white and carbohydrate in the form of rice were fed to cats there was no marked diminu tion in the size of the gallbladder This work had been presented before the Amer ican Association for the Advancement of Science an abstract of which appeared in the Anatomical Record With the publi cation of the method of Graham and Cole for visualization of the gallbladder follow ing the administration of the dye, Boyden s work on the cat was applied to man

Copher described the manner in which the gallbladder fills and empties He per formed the following experiments Gall bladders were emptied at laparotomy by manual compression and the cystic duct ligated After administration of the dye no shadow of the gallbladder was visible in the roentgenogram at the end of 24 hours At necropsy no bromine was found in the gallbladder The conclusion was therefore justified that the dye entered the gall bladder by way of the cystic duct

To prove that the dye also left by way of the cystic duct Copher carried out the following procedure After dense shadows of the gallbladder were obtained by the in jection of the dye the common duct was occluded The shadows persi ted until the death of the animal or until the opening of the common duct was re established In one dog, which lived for 11 days after the occlusion of the common duct the gall bladder shadow was still dense As a result of these experimental observations the con clusion was justified that the dye in the gallbladder left as it had entered namely, by way of the cystic duct

The emptying of the gallbladder by way of the common duct is also well demon strated in the experimental work of Walsh and Ivy [3] They obtained cholecystograms in 5 dogs following the administration of tetra iodophenolphthalein The common duct was then ligated After the daily inges tion of the fat meal the density of the gallbladder was maintained over a period of 2 weeks the entire duration of the observa tion It is therefore apparent that the gallbladder empties by way of the biliary passages Walsh and Ivy found that, in the dog the toxicity of the dye was not in creased in the presence of jaundice which they had produced artificially by occlusion of the common duct

That the fat meal administered for the emptying of the gallbladder need not come in direct contact with the papilla of Vater or be present within the duodenum in order to exert its effect was shown by Kalk and Nissen [4] They examined 5 patients who had had a Billroth II operation and 2 patients who had undergone gastro enter ostomy for benign pyloric stenosis in order to determine the effect of the fat meal on the emptying of the dye filled gallbladder They were able to obtain an adequate emptying response of the gallbladder under

the site of injection Reactions such as nausea vomiting diarrhea, headache and abdominal cramps, which occur after the administration by either method, are as a rule inconsequential, and even when marked rarely last more than a few hours The disadvantage of the oral method is that the degree of absorption of the dye cannot be as perfectly controlled as with the intravenous method When the dye is administered intravenously, the gallbladder begins to be visualized in 4 hours There is a gradual increase in the density, with a shadow exhibiting maximum concentration at 12 hours When administered by mouth, a considerably longer period of time elapses before the gallbladder can be visualized As a rule, a cholecystogram can be obtained in 12 hours, although, frequently, excellent concentration of the dye cannot be obtained until 18 hours after its ingestion

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complain of crampy pains and diarrhea and sometimes these are quite severe Burning on urination occurs very frequently in the male

The one outstanding advantage of priodax is that, as a rule none of the unabsorbed drug is present in the alimentary tract to obscure the field of vision In the occasional case when some of the drug is present, it is usually very small in amount Considerable gas, however is frequently present in the colon and the hepatic flexure often overlaps the gallbladder shadow In that event roentgen examination in the second oblique position may come to the rescue and enable us to isolate the unobscured shadow of the gallbladder I have found it necessary to employ this procedure very frequently in order to obtain a clear view of the gallbladder shadow The emptying of the gallbladder after the fat meal is studied in the same manner as with other media I have also observed just as in the case of these other media that in rare instances when there is a failure of visualization of the gallbladder shadow after the administration even of the double dose the drug may outline the gallbladder in the roentgenograms taken after the fat meal It is therefore a wise precaution to have the patient go through this procedure of taking the fat meal even in the case of a nonvisualized gallbladder shadow In every such case the gallbladder has been pathologic but it is obviously more satisfying to make a diagnosis of cholelithiasis when the nonopaque calculi are actually visualized as translucent areas than to depend entirely upon nonvisualization as the criterion

In the roentgen examination of the gallbladder the 11 by 14 size film is routinely employed at Bellevue Hospital as previously described in connection with radiography for the localization of biliary calculi (q v) This is important because of the marked variability in the position of the gallbladder It may be quite high up under the costal arch or well below the iliac crest It may extend laterally to a considerable degree or overlap the spine A large film therefore gives greater assurance that the dye filled gallbladder will not be cut off in the roentgenogram

Preliminary visualization of the gallbladder may also be made by fluoroscopic examination The advantage of this procedure is that not only may the gallbladder be visualized by scanning the entire field and determining its exact position before roentgen exposure but in addition one may readily determine the mobility of the viscus as will be emphasized later With the aid of the spot film device direct exposures may be made with or without the aid of compression

Routinely however roentgen exposures are made with the patient in the prone position and the rays are directed posteroanteriorly over the approximate region of the gallbladder area Superspeed screens a Bucky diaphragm and immobilization of the patient all aid in the sharp delineation of the cholecystogram After accurate localization of the exact position of the gallbladder the smaller area may then be brought more clearly into focus with the aid of a cone

Failure of visualization of the gallbladder after the administration of the dye may result from any of those factors which interfere with absorption by the liver or its ability to excrete the dye properly The following are among the important factors which may prevent visualization of the gallbladder

1 Vomiting of the dye obviously resulting in a deficient amount for absorption It is therefore of considerable importance to question the patient when the gallbladder is not visualized as to whether he retained the dye

2 Pyloric obstruction In such cases if the degree of obstruction is marked the dye is unable to pass through the pyloric ring into the small intestine where absorption takes place

3 Diarrhea When the dye having

these conditions In one case the fat meal was introduced by way of a tube which had entered the jejunum through the post operative stoma One hour after the fat meal, the dye filled gallbladder was empty They concluded that the expulsion reflex was induced in the upper jejunum In some cases however, they were unable to obtain as satisfactory a response of the gallbladder to the fat meal as under normal conditions

Cholecystography has been employed as a means of determining the effect of various stimuli on the emptying of the gall bladder [6] [7] The best method of emptying the gallbladder is found to be food rich in fat such as butter, cream or egg yolk Various drugs ordinarily considered to be of value in the emptying of the gallbladder had either no effect or were considerably less effective in causing the gallbladder to empty than the administration of the fat meal While the instillation of magnesium sulphate administered by duodenal tube produced a moderate diminution in the size of the gallbladder this response was no greater than that obtained from the administration of proteins and peptone by mouth [6]

TECHNIC OF CHOLECYSTOGRAPHY

So reliable have we found the oral method of cholecystography by the intensive or cumulative method of repeated doses that it is used routinely as the procedure of choice at Bellevue Hospital

The patient is kept on a rigid fat free diet during the course of examination The double dose method is employed in most cases The first dose is administered at 1 P M, shortly after lunch and the second dose at 7 P M shortly after the evening meal The patient then reports the following morning for examination Only if the evidence is inconclusive is the patient told to take an additional dose of the dye that evening and report again the next morning for further roentgen study

Following the oral administration of the dye, it is absorbed from the intestine and enters the liver, from which it is excreted into the intrahepatic ducts By way of the hepatic duct and cystic duct, the dye finally reaches the gallbladder itself Visualization of the dye filled gallbladder may then be noted 12 to 18 hours after the administration of the dye

In recent years β(4 oxy 3,5 diiodo phenyl) α phenylpropionic acid originally known as biliselectan has been extensively used for gallbladder visualization In this country it is known as priodax The original work in the production of this preparation was done by Dohrn and Diedrich [8] and its clinical value was confirmed by Kleiber [9] Grunke and Finger [30] Lauer Schmaltz[31] and Rating [3] Soon thereafter, the new preparation was favorably received in this country

The same precautions of a fat free diet are obviously equally important when the cholecystographic medium is priodax The dose ordinarily administered is 3 grams given in 6 tablets each containing 0 5 grams In most cases this amount of the drug is sufficient for satisfactory visualization of the gallbladder shadow At times, however, considerable improvement in the density of the shadow may be obtained when an additional 6 tablets (3 grams) is administered several hours later I have routinely followed this double dose procedure except in comparatively slender individuals and in children The first dose is administered shortly after lunch The patient is instructed to take 1 tablet every 2 or 3 minutes, so that approximately 15 minutes are consumed in the process The patient is encouraged to drink as much water as he wishes The second dose of equivalent amount is administered in the same manner shortly after dinner The patient then reports for roentgen study at 9 o clock the following morning He may have water or tea with sugar before arrival The roentgen examination is then conducted in the same manner as hitherto Reactions from the taking of the drug are quite common although never serious Patients not infrequently

larly from the pressure of a tumor of the pancreas upon the common duct

2 Shape The shape of the gallbladder varies from circular to pyriform It is influenced by the following factors (a) The tone of the gallbladder at the particular moment of examination may influence its shape thus the same gallbladder may exhibit a variation in shape due to differences in tone during the course of a single examination particularly as affected by the emptying process following the fat meal (b) Also as a result of the effort on the part of the gallbladder to empty itself waves may be present simulating peristaltic activity of the stomach although much less marked and more superficial in character (c) Variations in shape may be due to congenital anomalies This subject will be discussed more fully later

3 Position The gallbladder may show considerable variation in position To some degree this is influenced by the habitus of the patient Thus in the hypersthenic the gallbladder may be found high up under the costal arch In the hyposthenic and asthenic the gallbladder may be found below the intercrestal line The gallbladder may also overlie the vertebral column in such manner that it may escape detection unless this possibility is kept in mind The position of the gallbladder is also considerably influenced by the position of the patient Thus a film taken in the prone position may show the gallbladder to be considerably higher than when the film is taken with the patient in the erect position The gallbladder is also subject to displacement by extrinsic viscera and by gas in the hepatic flexure

4 Contour The gallbladder is normally extremely smooth in outline with no indentations or irregularities Even adhesions which may fix the gallbladder rarely produce any considerable degree of irregularity Congenital anomalies may alter its contour Irregularity of contour in the dye filled gallbladder may occur in carcinoma of this organ

5 Mobility This may be determined by the change in the position of the gallbladder brought about by variations in the position of the patient Fluoroscopy may be of considerable help in the demonstration of the degree of mobility of the gallbladder Another aid in determining fixation of the gallbladder is the simultaneous administration of barium by mouth and the introduction of barium by enema If the gallbladder is fixed to the duodenum or the hepatic flexure it may then be impossible to separate these regions manually under fluoroscopic control

6 Concentration of the dye A gallbladder that concentrates the dye poorly so that only a faint shadow is obtained should be viewed with suspicion This may be due to a deficient amount of dye entering the gallbladder because of the operation of the various factors previously described or even when the dye enters the gallbladder without hindrance there may be a disturbance in the concentrating mechanism within the gallbladder itself When the gallbladder wall is diseased there may be a failure in the absorption of water so that at no time is there a sufficient concentration of the dye to produce a normal shadow in the roentgenogram Every degree of qualitative variation may be found While a gallbladder shadow of poor concentration is significant of disease when the technic has been correctly followed there is still a moderately large percentage of error in that an apparently perfectly normal gallbladder may at times exhibit such evidence This is not nearly as diagnostic as complete failure of visualization of the gallbladder Unless the clinical history is strongly suggestive a poorly filled gallbladder is to be accepted only as tentative evidence of disease and further observation and checkup study are essential In this connection it is important to note that in some cases there may be a delay in the normal concentration of the dye so that a gallbladder radiographed between 12 and 18 hours after the administration of the dye may at a later examination

reached the small intestine, passes on with *abnormal* rapidity, there may be serious interference with sufficient absorption of the dye This point is particularly important, since the rapid emptying of the alimentary tract may fail to leave any evidence of dye in the colon which can be recognized in the film It is important that we instruct the patient not to take a cathartic prior to or during the period of examination

4 Fat in the diet If the patient has partaken of a fat meal shortly before the administration of the dye, it may act to empty the gallbladder before it has an opportunity of becoming sufficiently filled with the dye This is particularly important since fat leaves the stomach slowly If in *addition*, the stomach of the patient is hypotonic or atonic, there may be a sufficiently prolonged delay in the emptying of the stomach to enable fat to enter the intestine after the gallbladder has begun to fill with the dye, and this may cause it to evacuate its contents before a film has been taken

5 Disease of the liver In such cases the dye may be absorbed and enter the liver However, because of pathologic alterations of structure and function the dye may be retained for an abnormally long period of time or be excreted in an extremely slow manner A diseased liver may also fail to take up a sufficient amount of dye from the blood stream Therefore even without any serious interference with its excretory function, the liver may have an inadequate quantity of the dye within it The very small amount of the dye excreted by the liver may be insufficient after entering the gallbladder to produce roentgen evidence of its presence

6 Obstruction to the biliary ducts This may, for mechanical reasons prevent the *entrance* of the dye into the gallbladder This is particularly true of malignancies involving the hepatic or cystic ducts This will also occur in the presence of inflammation of the cystic duct or of impaction by a stone Also, if a stone in the gallbladder is present near the mouth of the cystic duct, or if many stones choke up the gallbladder so that the dye cannot enter, there will be failure of visualization *of the gallbladder In addition, if the gall*bladder is filled with mucus or pus, its proper filling with the dye may be interfered with

7 Disturbance in the concentrating function of the gallbladder After the dye has actually reached the gallbladder, it may still fail to be visualized roentgenologically because of such disturbance As a result of disease of the gallbladder wall there is a failure of absorption of water, with consequent inability to concentrate the dye sufficiently to cast a shadow in the roentgenogram *Bollman*, Mann and *DePage*[33] produced a specific cholecystitis in dogs by intravenous injections of chlorinated soda They were thereby able to demonstrate an impairment in the concentrating mechanism of the gallbladder

Filling the gallbladder with the dye enables us to determine its size, shape position, and degree of concentration, as well as its motor behavior

1 Size The size of the gallbladder may vary considerably within the range of the normal It will also vary somewhat depending *on the position of the patient and the relation of the viscus to the angle of the* rays Since the gallbladder is a three-dimensional organ and is projected on a two dimensional film, there may be considerable variation in the roentgenogram as compared with the actual size of the gallbladder itself, depending upon its actual anatomic position in relation to the rays It may be foreshortened or appear as if cut *in cross sec*tion Its size will also vary with the period of examination, depending upon the degree of filling with the dye The size will likewise be influenced by the tone of the gallbladder, and this will depend to some degree upon the habitus of the individual The gallbladder may be enlarged to an abnormal degree as a result of obstruction particu

larly from the pressure of a tumor of the pancreas upon the common duct

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appear perfectly normal Rarely, a gall bladder shadow which either fails to be visualized or is poor in quality may, after the fat meal exhibit a normal appearance This may in part at least, be due to the evacuation of the less dense dye at the upper pole of the gallbladder, so that the denser dye at the lower pole may then fill up the organ In addition, there has been a further lapse of time during which there may have occurred an increased concentration of the dye still remaining in the gall bladder

the gallbladder, a stone may be seen through the thin layer of dye which remains Taking a film with the patient in the erect position may al o be helpful The calculi sinking to the lower pole may then produce negative shadows in this region In addition, stones may gather in an intermediate position between the denser dye at the lower pole of the gallbladder and the more fluid, less dense area above it

Åkerlund[31] recommended the upright position with graded compression in the roentgen examination of the gallbladder

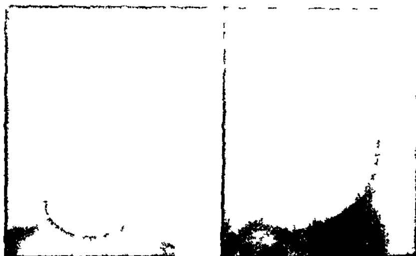


Fig 571 (A *Left*) Normal cholecystogram (B *Right*) Normal cholecystogram

7 Negative shadows within the gallbladder Of great clinical significance is the demonstration of negative shadows within the dye filled gallbladder In this manner nonopaque calculi may be readily diagnosed which otherwise would escape detection even with the most careful technic While calculi may thus be diagnosed an apparently homogeneously filled gallbladder may in a small percentage of cases also contain stones which do not produce translucent areas and are actually obscured by the dense shadow of the dye

A number of aids may be employed to show the pre ence of calculi under such conditions After the fat meal, as the dye leaves

for the demonstration of small calculi In the erect position such calculi sink to the fundus of the gallbladder the position of which will remain constant However some gallstones may apparently remain so adherent to the mucosa as to be unaffected by change of position In five cases Akerlund showed that small calculi were suspended horizontally in the middle of the gallbladder shadow He explained this by assuming that the bile at the base of the gallbladder is heavier than that in the upper layers As a result the calculi remain stratified between these two layers Beautiful examples of this phenomenon are shown in the cholecystograms which accompany his

article Bermond[35] Brailsford,[36] Bernstein[37] Prevot[38] and Ettinger[39] verified the work of Akerlund and demonstrated the horizontal layer of calculi in the cholecystogram taken in the erect position In some cases Bernstein was able to demonstrate two superimposed layers of biliary calculi

Gas in the duodenum or in the hepatic flexure may overlap the dye filled gall bladder in such manner as to simulate the negative shadows of gallstones Differentiation is ordinarily simple The continuity of the gas with the rest of the intestine may be traced At times an isolated gas bubble may be seen partly overlapping the gallbladder but also in part extending beyond its confines In different films such gas may be found to occupy different positions in relation to the gallbladder Changing the position of the patient in relation to the angle of the rays will also help to separate the gallbladder from overlying areas The appearance of the gallbladder during contraction following the administration of the fat meal will frequently aid in such differentiation As the cholecystogram diminishes in size the confusing shadow may then be shown to be beyond the confines of the gall bladder itself In the event of persistent doubt, examination after a cleansing enema or a second examination of the gallbladder will solve the difficulty

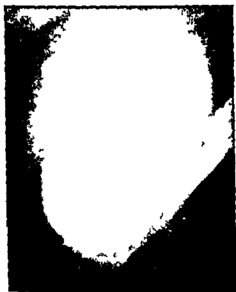




Fig 572 (*Left*) Normal cholecystogram under the right costal arch
Fig 573 (*Right*) Low position of the gallbladder

Collins and Root[40] have recommended the administration of 1 cc of pitressin in order to eliminate confusing shadows in the alimentary canal which obscure detailed visualization of the cholecystogram Owing to the drop in blood pressure which frequently occurs from the use of pitressin, however it is contraindicated in hypertension or in those whose systolic pressure is below 100 mm of mercury Scheibel[41] also demonstrated the excellent value of pitressin for eliminating gas in the colon overlapping the gallbladder He recommended the injection of 10 units of pitressin ½ hour before examination

The most effective method in my experi

ence, for the separation of the gallbladder shadow from overlapping gas or other extrinsic areas is examination in the second or left oblique position The angle of obliquity should be about 45 degrees and the cone focused over the gallbladder so that the lateral margin of the rim of the cone is tangential to the right border of the abdominal wall The exact degree of obliquity best suited to isolate the gall bladder shadow will obviously have to be determined in each particular case

In addition to the elimination of overlapping shadows examination in the second oblique position will occasionally give a better idea of the size of the gallbladder since it may be foreshortened when the viscus is examined at an angle of 90 degrees



Fig 574 (A *Left*) Low position of the gallbladder overlapping the spine (B *Right*) Same patient as is shown in (A) Appearance after a fat meal



Fig 575 (A *Left*) Position of the gallbladder 16 hours after administration of the dye (B *Right*) Same patient as is shown in (A) Eighteen hours after administration of the dye Note the marked change in the position of the gall bladder

8 Emptying of the gallbladder After visualization of the gallbladder a fat meal

is administered This may consist of 3 eggs or 2 eggs with bread and butter and a glass of half milk and half cream Ordinarily the patient is examined ½ hour later at which time the normal gallbladder may exhibit a diminution in size of about 50 per cent At times however the gallbladder may cystic duct such fractional studies may be important

Delayed emptying of the gallbladder occurs normally if the patient is starved or lives primarily on carbohydrates Another possibility is that new dye enters the gallbladder as it empties so that it remains



Fig 576 (A *Left*) Note the overlapping of the lower pole of the gallbladder by gas (B *Right*) Same patient as is shown in (A) Examination in the left oblique position The lower pole of the gallbladder is now free of overlapping gas



Fig 577 (A *Left*) Note small rounded translucent areas in relation to the gallbladder shadow (B *Right*) Same patient as is shown in (A) Examination in the left oblique position The gallbladder shadow is now free of any overlapping gas

empty completely or if atonic may show only a slight diminution in size When a stone is suspected in an apparently normal looking gallbladder films may be taken at 10-minute intervals In this manner a negative shadow may be noted before complete emptying of the gallbladder occurs and when the dye has been thinned permitting the stone to shine through as a negative shadow Also for the visualization of the filled for an abnormally long period of time or there is a reabsorption of the dye after elimination from the gallbladder which then again finds its way into the viscus Functional derangements such as spasm of the sphincter of Oddi with a normally contracting viscus may possibly play a role in delayed emptying of the gall bladder in the absence of organic disease Marked narrowing of the cystic duct,

though permitting the entrance of the dye, may interfere with its free egress

In the presence of organic disease of the gallbladder wall, the dye may in some cases, nevertheless, be sufficiently concentrated to show as a cholecystogram Because of the rigid character of the wall, the gallbladder may be unable to expel the dye and it may therefore remain roentgenologically visible for an abnormally long period When the common duct is obstructed as the result of a tumor such as of the pancreas the dye may enter the gallbladder by way of the *cystic duct but will be unable to empty* itself through the common duct The size of the gallbladder shadow therefore will remain unaffected by the fat meal for an abnormally long period

6 Exclusion of extrabiliary shadows Not only is cholecystography of value for the demonstration of derangements of structure and function due to causes within the biliary apparatus itself, but it also possesses considerable diagnostic value by excluding shadows of a suspicious character which might conceivably have been produced by biliary calculi or calcifications of the gallbadder wall In this manner a normal cholecystogram may *prove beyond* a doubt the fact that the calcific shadow is outside the confines of the gallbladder itself On the other hand it may also show that the calcific shadow is actually within the gallbladder by proving that it is present within the confines of the cholecystogram



Fig 578 (A *Left*) Gas overlapping the lower pole of the gallbladder (B *Right*) Same patient as is shown in (A) Examination in the left oblique position The gallbladder shadow is now free of overlapping gas



Fig 579 (A *Left*) Note superimposed area at the lower pole of the gallbladder shadow (B *Right*) Same patient as is shown in (A) Appearance of the gall bladder shadow in the second oblique position The area at the lower pole of the gallbladder (A) was due to the projection of the lower pole upon the rest of the gallbladder shadow

Illustrative Cases Figure 571 shows examples of normal cholecystograms

The gallbladder may vary considerably in position within the range of the normal, an important factor being the habitus of the individual Figure 572 shows a normal gallbladder up under the right costal arch It is of globular shape



Fig 580 (A *Left*) Cholecystogram showing two calcific areas in the region of the lower pole (B *Right*) Same patient as is shown in (A) Examination in the left oblique position At this time the calcific areas are outside the confines of the gallbladder shadow



Fig 581 (A *Left*) Appearance of the gallbladder shadow Examination in the postero anterior position at an angle of 90 degrees (B *Right*) Same patient as is shown in (A) Appearance of the gallbladder shadow in the second oblique position thereby showing the real size of the gallbladder

The gallbladder may also be very low in position (Fig 573) At times it may actually overlap the spine as illustrated in Figure 574A Note also the relative change in the shape and position of this gallbladder after the fat meal as well as the increased concentration of the dye at this time (Fig 574B)

Another factor which may cause considerable variation in the position of the gallbladder is the pressure of the gas distended colon This is well illustrated by the changes not only in position but also in contour, of the gallbladder in the same patient as the result of such pressure Figure 575A 16 hours after administration of the dye, shows the gallbladder under the costal arch almost transverse in position above the gas distended hepatic flexure Figure 575B at 18 hours, shows the gallbladder elongated and essentially vertical

The left oblique position in the examination of the gallbladder is of considerable value for a number of reasons

1 It is the most practical procedure for the elimination of overlapping gas as illustrated in Figure 576A and B In Figure 576A the lower pole of the gallbladder shadow is

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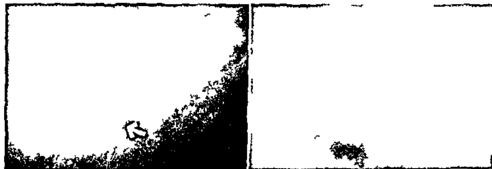


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Fig 581 (A *Left*) Appearance of the gallbladder shadow Examination in the postero anterior position at an angle of 90 degrees (B *Right*) Same patient as is shown in (A) Appearance of the gallbladder shadow in the second oblique position thereby showing the real size of the gallbladder

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overlapped by gas This may prove to be confusing in two ways First the gas may simulate the translucencies due to actual calculi within the gallbladder itself Secondly, even when it is reasonably certain that the shadows are the result of intestinal gas they may obscure a calculus present in that particular area of the gallbladder Figure 576B shows the appearance of the gallbladder shadow in the left oblique position

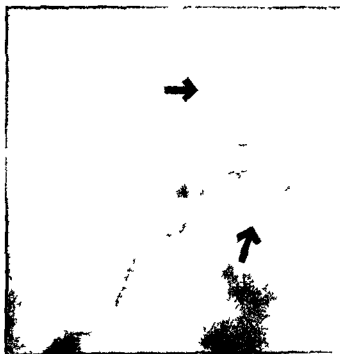


Fig 582 Cholecystogram including the cystic duct the hepatic duct and the common duct

It is now free of the overlapping gas and is normal in appearance This point is further illustrated by Figure 577A and B Figure 577A (postero anterior position at 90 degrees) shows a number of small translucent areas in the region of the gallbladder shadow That these areas are of extrinsic origin and due to overlapping gas is shown by the appearance in Figure 577B (second oblique position) The value of the procedure may also be seen in a comparison of Figures 578A and 578B

2 The second oblique position may eliminate suspicious shadows in the cholecystogram due to foreshortening Thus in Figure 579A there is a circular area apparently within the confines of the lower pole of the gallbladder shadow Figure 579B (second oblique position) shows the gallbladder shadow to be normal In Figure 579A the lower pole of the gallbladder had become superimposed on the rest of it

3 The second oblique position may be important in order to show that calcific areas which might conceivably be within the confines of the gallbladder shadow are actually extrinsic to it Figure 580A shows two small calcific areas in the region of the cholecystogram Figure 580B (second oblique position) shows that they are of extrinsic origin

4 Because of foreshortening the second oblique position may give a better idea of the actual size of the gallbladder Figure 581A shows the size of the gallbladder in the conventional postero anterior position

at an angle of 90 degrees Figure 581B (left lateral position) shows the actual length of the gallbladder In addition, it is not over



FIG 583 (A Left) Faint visualization of the gallbladder shadow after a single dose of the dye (B Right) Same patient as is shown in (A) Note the clear visualization of the gallbladder shadow after administration of two additional doses of the dye The gallbladder is packed with stones

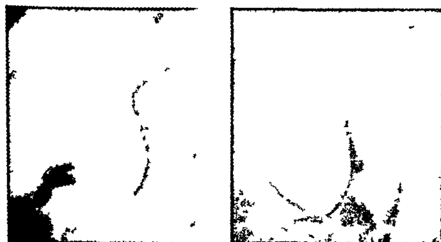


FIG 584 (A Left) Apparently normal cholecystogram No negative shadow is noted (B Right) Same patient as is shown in (A) This gallbladder in addition to showing excellent concentration of the dye shows a faint but definite translucent area within it One large and several small calculi were found at operation

lapped by gas at this time as in the previous observation

In some cases the cystic and common ducts may be visible in addition to the gallbladder The best time to visualize the cystic and even the common duct is shortly after the fat meal At this time the gallbladder may be caught in the very act of emptying itself of its contents, and the entire length of the biliary duct may then be visualized (Fig 582) This examination

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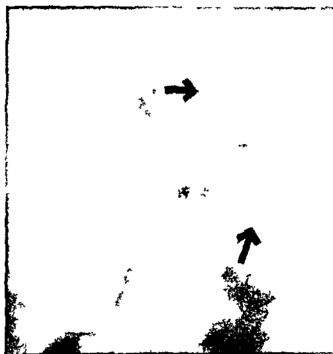


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cellent concentration of the dye and (3) even though one translucent area corresponding to a calculus was finally demonstrated several other small calculi found at operation could not be demonstrated even by this second examination It is possible therefore for a calculus to be present and yet go unrecognized and for the gallbladder at the same time to appear objectively normal on roentgen examination

The presence of many calculi in the gallbladder may not only be associated with a normal concentrating mechanism but such strable in the erect position is shown in Figure 589

A rare type of abnormality is the star shaped translucent area in the center of a gallstone [43 44 45 46]

In some cases the calculus and the central branched translucent area may both be visible In rare instances only the central translucency may be noted This is therefore a point of significance in the roentgen diagnosis of cholelithiasis in the absence of the demonstration of the calculus itself





Fig 586 (*Left*) Large translucent areas produced by calculi
Fig 587 (*Right*) Translucent areas within the gallbladder shadow produced by calculi

a gallbladder may also exhibit an excellent response to the fat meal Thus Figure 585 A shows a dye filled gallbladder containing many calculi After the fat meal there was considerable reduction in the size of the gallbladder At this time due to partial emptying of the dye the entire gallbladder appeared to be packed with stones showing as numerous small translucent areas (Fig 585 B)

Characteristic examples of the translucent areas produced by calculi within the dye filled gallbladder are shown in Figures 586 587 and 588

The layer of calculi occasionally demon

To determine the cause of this translucent area Kommerell and Wolpers[44] broke up the stones under water thereby releasing a gas which was odorless and on analysis yielded 0 5 per cent oxygen and 6 to 7 5 per cent carbon dioxide the rest being nitrogen Because of the gas within its confines the calculus was able to float not only in bile but even in distilled water They believed that the gas was the result of organic decomposition possibly due to bacterial activity

The appearance of the star shaped translucent areas within the confines of gallstones is shown in Figure 590

was made 15 minutes after the fat meal A finding of this nature is not uncommon, and I am not convinced that it has any clinical significance It is due entirely, I think, to good fortune in "catching" the gallbladder during the act of expulsion of its contents

Ivy[4] believed that cholecystographic visualization of the hepatic duct associated with pain after the ingestion of the fat meal might be considered as suggestive evidence of biliary dyskinesia

Illustrative Cases The value of repeated cumulative doses of the dye in the demonstration of the gallbladder is illustrated by the following surgically controlled case

B K female aged 53 (Fig 583A and B) Eighteen hours after a single dose of the dye only a very faint shadow of the gallbladder was visible (Fig 583A) Two additional doses of the dye were then administered Eighteen hours after the third dose the gallbladder shadow was well visualized and numerous translucent areas were present within it (Fig 583B) The diagnosis of cholelithiasis was verified at operation

Well concentrated dye in the gallbladder may conceivably obscure a calculus within it This point is illustrated by the following case

S P male aged 43 This patient had his first attack of colicky pain in the right lower quadrant 1 year before his admission to the hospital Previously he had had gastric distress for about 3 years Since his first attack he had at least four mildly severe attacks At one time he was jaundiced

Physical examination revealed tenderness in the right upper quadrant No masses were palpable

At operation the gallbladder showed a few adhesions to the omentum which were freed easily The gallbladder was of fairly normal color It contained one large and several small stones No stones were found in the common hepatic or cystic ducts

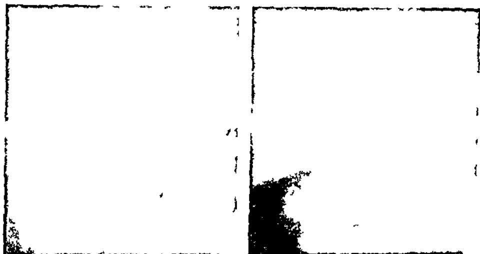


Fig 585 (A *Left*) Gallbladder with calculi Many are obscured by the dye (B *Right*) Due to partial emptying of the dye the entire gallbladder appears to be filled with stones

Roentgen examination about a year prior to operation (Fig 584A) showed an apparently normal gallbladder with excellent concentration of the dye No negative shadows were noted at any time during the examination including the film after the fat meal Because of the persistence of clinical symptoms re-examination was carried out about 1 year later (Fig 584B) At that time roentgen examination in addition to showing a gallbladder with excellent concentration of the dye also showed a faintly outlined translucent area within it A diagnosis of cholelithiasis was then made which was verified at operation

There are a number of interesting points brought out by the above case (1) a gallstone can be obscured by the dye (2) a gallbladder containing calculi may show ex

cellent concentration of the dye and (3) even though one translucent area corresponding to a calculus was finally demonstrated several other small calculi found at operation could not be demonstrated even by this second examination It is possible therefore for a calculus to be present and yet go unrecognized, and for the gallbladder at the same time to appear objectively normal on roentgen examination

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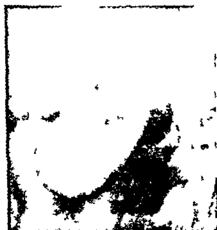




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CHOLECYSTOGRAPHIC DIAGNOSIS OF TUMORS OF THE GALLBLADDER

The diagnosis of benign tumors of the gallbladder, as shown by Kirklin,[47] may be made on the basis of the following roentgen characteristics The defect is oval or round and has a diameter usually less than 0 5 cm The defect may be single, but occasionally two or three discrete areas may be present Particularly suggestive is a marginal localization of the defect *The relative situation* remains unchanged The favorite situation of adenomas is in the fundus and they are apt to be larger than papillomas Such benign tumors as papillomas or adenomas may sometimes best be shown during the contracting phase after a fat meal Unlike tumors of the gallbladder small cholesterin gallstones may alter their relative position from time to time Also during contraction of the gallbladder they may become closely grouped together Hefke[48] and Moore[49] corroborated these roentgen findings in tumors of the gallbladder





Fig 588 (*Left*) Translucent areas within the gallbladder shadow produced by calculi

Fig 589 (*Right*) Examination of the gallbladder in the erect position showing translucent areas produced by a layer of calculi (Dr Ettinger)

Illustrative Case I am indebted to Dr B R Kirklin for the privilege of including this case of papilloma of the gallbladder The roentgenogram (Fig 591A) exhibits a small translucent area near the inner border of the gallbladder This was present at 14 hours and 16 hours after the administration of the dye and 1 hour after the fat meal The accompanying photograph of the specimen (Fig 591B) shows the appearance of the papilloma which was found at operation and which corresponds to this translucent defect

Cholecystographic visualization of a carcinomatous gallbladder is extremely rare The following case is the only one in my personal experience

M S, male, aged 70 During the preceding 4 weeks the patient had attacks of sharp right upper quadrant pain radiating to the back Physical examination revealed an icteric tinge to the conjunctivae The liver edge was palpable 1½ inches below the costal margin While under observation jaundice became more marked and he developed ascites

At operation 3 200 cc of fluid were aspirated The surgeon stated that the pancreas was stony hard with metastatic spread to the gastrohepatic omentum along the common bile duct the portal vein and the duodenohepatic ligament The entire gallbladder wall was involved and stony hard Numerous metastatic nodules were present in the liver A biopsy was taken from the gastrohepatic omentum

The pathologic diagnosis was carcinoma probably secondary to carcinoma of the biliary tract

The autopsy findings were as follows

'Gallbladder and bile ducts The gall

bladder is hard and nodular and contains two stones near the fundus One stone measures 2 cm in its greatest diameter and the other a round stone measures 1 cm in diameter There is a growth in the fundus of the gall bladder which is irregular thickened to 1 cm and has a gelatinous consistency The common bile duct is completely constricted at the pylorus by the mass of tumor matted together in the superior gastric and subpyloric nodes It is this mass of tumor tissue that has invaded the head of the pancreas by direct extension On sectioning this mass the portal vein is found to be completely surrounded and compressed by the tumor and there is a thrombus occluding its lumen This mass has extended into and invaded the first part of the duodenum

Gallbladder Section through the gallbladder reveals a large part of the wall of the gallbladder to be thickened and replaced by tumor tissue This consists of loose aggregates of large irregularly shaped and staining mononuclears and multinuclear giant cells with dark staining nuclei The individual cells are separated by strands of loose connective tissue The tumor mass is heavily infiltrated with acute and chronic inflammatory cells It consists of interlacing papillomatous masses of tumor tissue with a central connective tissue stalk In another portion of the gallbladder the tumor takes on a glandlike appearance At one portion of the gallbladder the wall is markedly thickened and the outer portion consists entirely of connective tissue while the inner portion is composed of tumor tissue which appears as numerous confluent pools of mucin separated by interlacing bands of hyalinized connective tissue stroma Suspended in the mucin are tumor cells and polymorphonuclear leucocytes The blood vessels are markedly congested and there are areas of hemorrhage into the tissue The whole gallbladder is heavily infiltrated with chronic and acute inflammatory cells

The final diagnosis was carcinoma of the gallbladder with secondary invasion of lymph nodes and pancreas

Cholecystography (Fig 592) revealed an unusual appearance of the gallbladder shadow The contour was irregular and there was a peculiar distribution of the contrast substance throughout the gallbladder shadow It was somewhat mottled and lacked homogeneity There were some vaguely outlined translucent areas within the confines of the gallbladder shadow

Although it was quite obvious that we were dealing with an unusual condition of the gallbladder the exact nature of the pathologic process was not clearly understood until autopsy revealed the primary malignant lesion of the wall of the gallbladder Apparently in spite of the extensive and serious disease of the wall the gallbladder was able to concentrate the drug (priodax) sufficiently to cast a shadow in the roentgenogram The irregular configuration and the mottled char-

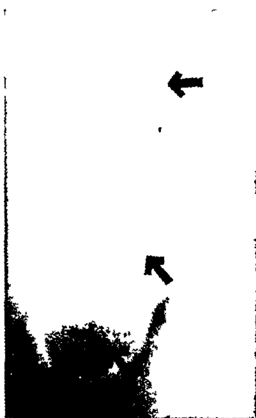


Fig 590 Star shaped translucent areas within calculi (Dr Ortmayer)

acter of the shadow are apparently due to the malignant infiltration of the wall

ADMINISTRATION OF THE DYE IN THE PRESENCE OF JAUNDICE

My personal experience with the use of the dye in the presence of jaundice leads me to the opinion that the procedure is essentially innocuous and in a case where an obstructive type of jaundice is suspected the method may yield data of considerable

aid in differential diagnosis This experience regarding the safety of administering the dye in the presence of jaundice for purposes of differential diagnosis has been confirmed by other observers

Fried and Whitaker[9] *showed in their* experiments on dogs that even when the liver has been moderately damaged by chloroform as an anesthetic this is no con traindication to the use of sodium tetra



Fig 591 (A *Left*) The gallbladder at 14 hours shows a small translucent area within it Compare with (B) (B *Right*) Same patient as is shown in (A) Papilloma of the gall bladder found at operation

iodophenolphthalein The dye may be safely used in the human being even in the pres ence of obvious liver damage particularly since the dose ordinarily used in patients is one fourth that used in the experiments on dogs with known extensive disease of the liver In cases in which an extreme degree of liver damage was present, however, there was a failure of visualization of the gall bladder

That severe liver impairment may pre vent visualization of the gallbladder was further corroborated by the work of Nord and Blomstrom and Sandstrom Nord[1] demonstrated that it was often impossible *to obtain a normal cholecystogram in the* presence of an acute hepatitis, particularly when the degree of icterus was increasing There appeared to be a parallelism between the ability to eliminate the coloring sub *stance of the bile from the blood and ability* to eliminate the dye Blomstrom and Sand strom showed that a retention of brom sulfalein of over 40 per cent apparently precluded the possibility of obtaining a cholecystogram due to impaired liver func tion

That the administration of the dye in the presence of jaundice is ordinarily an innoc uous procedure is shown by the following reports Rudisill[3] carried out cholecysto graphic examinations on 10 jaundiced pa tients without ill effects to any of them In 8 of these cases the clinical diagnosis was catarrhal jaundice and in 7 there was nor mal visualization of the gallbladder The conclusion appeared justified that not only was there no danger in administering the dye in the presence of jaundice, but that it was of practical diagnostic value

Foley[34] demonstrated the freedom from danger of administering the dye to jaundiced patients either by the oral or intravenous route In 16 cases of nonsurgical jaundice the gallbladder was not visualized Jacobi[35] visualized the gallbladder in 7 out of 8 cases of toxic hepatitis without deleterious effect Foote and Carr[d] similarly showed that in a large number of cases of hepatitis visualization of the gallbladder was possible by the slow intravenous administration of the dye well diluted in 10 per cent glucose solution The innocuous nature of the dye therefore even in the presence of jaundice or actual hepatitis is convincingly demonstrated by these experiences

Moreover the method may be used for differential diagnosis If in the presence of jaundice the gallbladder fills and empties normally hepatitis is probably the cause Should cholecystography reveal a gallbladder shadow which however fails to empty after the fat meal the jaundice is probably obstructive in nature

Illustrative Case The diagnostic value of cholecystography in the presence of jaundice is illustrated by the following case

J B male aged 61 One month before his admission to the hospital the patient noticed that his skin had become yellow This yellow color increased and was accompanied by severe itching He had no pain The patient said that his stools were white He had no chills fever vomiting or ascites He had lost 15 pounds during the preceding month

Physical examination revealed a generalized icterus Examination of the abdomen showed the liver palpable five fingers breadth below the costal margin it was not tender and was smooth At the liver edge there was a firm nontender mass which moved with the liver on respiration The jaundice gradually became more intense

Operation revealed a carcinoma of the head of the pancreas about 3 inches in diameter stony hard and not adherent The gallbladder was enlarged to about 3 inches in diameter at the fundus and was adherent to the liver which was free of metastasis The lymph nodes in the root of the stomach and liver were enlarged and a large amount of inspissated bile was found in the gallbladder Cholecystgastrostomy was performed

The report of the autopsy was The head of the pancreas feels enlarged and of extremely firm consistency It encircles the termination of the common bile duct The gallbladder is dilated but appears collapsed and emptied A stoma admitting the index finger is found communicating between the pyloric portion of the stomach and the gallbladder The gallbladder wall appears thick



FIG 592 Carcinoma of the gallbladder Note the unusual fuzzy appearance of the cholecystogram as well as the superficial irregularity of the contour

ened The cystic duct and common bile duct down to a point 4 cm from the papilla of Vater are markedly dilated the common bile duct reaching a circumference of 3 5 cm At a point about 4 cm from the papilla of Vater the common bile duct appears narrowed to a circumference varying from 5 to 8 mm This narrowing occurs abruptly and corresponds to the entrance of the common bile duct into the head of the pancreas

Microscopic examination of the pancreas reveals a scirrhous adenocarcinoma The final diagnosis is carcinoma of the head of the pancreas stenosis of the pancreatic and common bile ducts

Cholecystography revealed a very large gallbladder with somewhat diminished density of the shadow (Fig 593A) Two hours after the fat meal there was no diminution in the size of the gallbladder shadow (Fig 593B) The reason for this marked increase in the size of the gallbladder shadow may be readily

understood from the findings at autopsy, showing a carcinoma of the head of the pancreas which had obstructed the common bile duct There was secondary dilatation of the gallbladder as a result of this obstruction Such obstruction of that portion of the common bile duct passing through the head of the pancreas does not prevent the entrance of the dye into the gallbladder by way of the cystic duct For this reason the large gallbladder is clearly demonstrated following the administration of the dye However, owing to the marked obstruction of the distal portion of the common bile duct, the dye was unable readily to

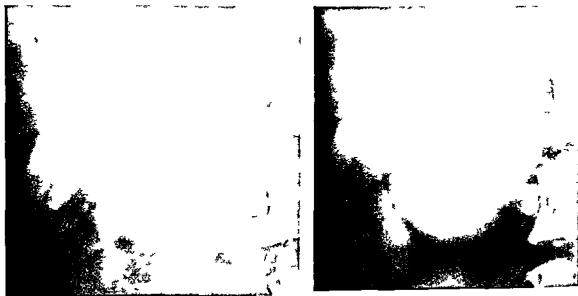


Fig 593 (*A Left*) *Cholecystogram in a case of carcinoma of the head of the pancreas with jaundice Note the increased size of the gallbladder shadow* (*B Right*) Same patient as is shown in (A) Failure of the gallbladder shadow to diminish in size after the fat meal is due to obstruction of the common duct

leave the gallbladder after the fat meal The gallbladder therefore remained filled with the dye and showed no evidence of any diminution in size

This case demonstrated two points (1) that the administration of the dye is not ordinarily contraindicated in the presence of jaundice, and (2) that cholecystography in the presence of jaundice may be of diagnostic value

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61

Congenital Anomalies of the Gallbladder

The importance of a knowledge of the occurrence of aberrant biliary vesicles is well described by Boyden [1]

> In view of the new methods employed in visualizing the human gall bladder without resort to operation it has become more than ever necessary to provide a comparative and embryological background against which congenital anomalies of the human gallbladder may be interpreted and classified It is now possible to arrange aberrant hepatic vesicles into four genetic groups all of which originate early in embryonic life namely, cleft gallbladders comprising forms which originate by initial subdivision of the primary cystic diverticulum of the embryo diverticular bladders distinct vesicles or subordinate lobes arising as buds from the neck of the embryonic gallbladders supernumerary vesicles derived from either hepatic cystic or common bile ducts and trabecular bladders vesicular outgrowths of liver trabeculae bordering the fossa vesicae felleae

Partial or complete duplication of the gallbladder though not uncommon in the cat calf, sheep and pig is rare in the human being In a series of 19 000 cadavers and patients examined roentgenologically the condition was found five times

Dilatations of the bile duct at the ampulla simulating an accessory gallbladder have been noted in the otter walrus seal and elephant (Mentzer)

DOUBLE GALLBLADDER

Gross[2] made a detailed study of 148 cases of congenital anomalies of the gallbladder of particular interest not only from a clinical but also from a roentgenologic point of view He collected 28 examples of double gallbladder with two separate gallbladder cavities and two separate cystic ducts If the two gallbladders lying next to each other are invested by a common peritoneal coat the true nature of the condition may escape the attention of the surgeon The accessory gallbladder may however be present in other regions such as the under surface of the left lobe of the liver Boyden included six cases of double gallbladder in the human described in the older literature which are not recorded in the series by Gross Double gallbladders apparently originate from a persistence of supernumerary buds normally present in embryonic life and arising from the extrahepatic ductal system

Bilobed gallbladders have been reported in man [3] They are frequently present in the cat and occasionally in the giraffe The bilobed gallbladder common in many of the higher vertebrates is rare in man In one type the external appearance is normal, but on opening the viscus there is a longitudinal septum which divides it into two chambers both drained by a single duct In the second type the gallbladder is bilobed, the two parts completely separated but fused near the cystic duct

Sherren [4] in 1910, operated on a woman 25 years old, who gave a long history of attacks of right upper quadrant pain radi

ating to the right shoulder and relieved by belching On opening the abdomen he found *two complete gallbladders joined only along* a narrow portion of their circumference Each gallbladder possessed its own cystic duct The specimen is in the museum of the Royal College of Surgeons of England Sherren was able to find a record of only one similar case, found at autopsy and reported by Purser in 1886

The case of double gallbladder reported by Nichols is very interesting Roentgen examination disclosed two distinct rows of calculi in the gallbladder region At operation *a double gallbladder was found, with* two distinct pouches joined by connective tissue Each gallbladder had its own cystic duct and contained stones

Croudace[6] found two gallbladders at operation *which had not been discovered* cholecystographically The cystic duct of one of the gallbladders penetrated deeply into the liver substance where it could not be traced to its conclusion One of the gallbladders was of average size and was normal The second one was half the size of the other and contained twenty nine small gallstones

The case described by Gross in 1936 showed a double gallbladder at autopsy The patient a child aged 3 had died of bronchopneumonia *No symptoms had been* present at any time referable to the biliary tract Additional cases of double gallbladder were reported by Braun [7] Hayes[8] and Cave [9]

ANOMALIES OF THE DUCTS

The following anomalies of the ducts have been reported Congenital absence and stricture of the cystic duct reduplication of the cystic duct and various abnormal *sites of termination, into the left hepatic* duct into the common duct and even into the stomach and duodenum In the case of the hepatic duct there have been reported congenital absence congenital atresia cystic *hepatic ducts draining* from the liver into the gallbladder or into the cystic duct and multiple hepatic ducts

HOURGLASS GALLBLADDER

One of the earliest descriptions of hourglass gallbladder was by Osler[10] in 1881 Hartmann [11] in 1891, described cases of *hourglass formation of the gallbladder* with a gallstone in each compartment Because of the fact that calculi were present in the cases described by Osler and Hartmann, it is difficult to state whether the deformity was *really* of congenital origin

Triloculation of the gallbladder may also occur but it is very rare

THE 'PHRYGIAN CAP

*An interesting and not infrequent anom*aly of the gallbladder is that of apparent angulation of the proximal portion of the gallbladder upon itself This anomaly was described by Bartel[1] as the Phrygian cap *Although the deformity is undoubtedly of* congenital origin, it is noteworthy that in the first three cases described by Bartel he found cholesterin stones to be present The congenital nature of the anomaly was shown *clearly by Boyden*[13] and *Cesarini* [14]

When a gallbladder of this type is examined anatomically one may find two types of abnormality In one type the gallbladder appears externally normal the indentation being obscured by the serosa *(retroserosal type)* In the second type the serosa also partakes in the indentation When the gallbladder is opened up one may then note the septum reaching into the lumen of the gallbladder and thereby producing various *forms* of biloculation When the septum is studied histologically there is no evidence of inflammation

The cholecystographic appearance corresponds to the anatomic description The indentation produced by the septum may occasionally be noted as a sharply outlined narrow translucent shadow extending for a variable distance into the *lumen of the dye* filled gallbladder The portion of the gallbladder proximal to the indentation appears angulated upon the rest of it like a cap In some cases only the indentation in the ex

ternal contour produced by the septum may be noted This may be due to the fact that the thin septum is obscured by the dye or that the cap partially overlaps the rest of the gallbladder After the fat meal the entire gallbladder empties normally without the septum exercising any restraining influence upon the evacuation of the dye in the angulated area Both in the ability of the gallbladder to concentrate the dye and in its motor activity it behaves in an entirely normal manner The practical clinical importance of recognizing the congenital nature of this deformity is that one should not be misled into assuming that the deformity is due to inflammatory pathology either extrinsic or intrinsic to the gallbladder itself On the other hand it must be quite obvious that a congenital anomaly of this kind does not necessarily make the gallbladder immune to the development of disease within it and as Bartel originally stated and as I have personally noted gall stones may be present within a gallbladder of this kind

The Phrygian cap type of anomaly can readily be distinguished from a hook shaped anomaly of the gallbladder by the fact that in the latter the contour is uninterrupted by any evidence of an indentation

DIVERTICULUM OF THE GALLBLADDER

A rare anomaly of the gallbladder is that of congenital diverticulum Courvoisier[15] found records in the literature of 28 cases of diverticulum of the gallbladder and credited von Heister in 1717 with having given the first description of such a case The appearance has been describ d roentgeno logically by Barsony and Friedrich[16] Camplani[1] Vastine[15] Hartmann[19] and Porta and Pilotti

The cholecystogram in this condition shows a rounded saccular area which appears to be tied off from the rest of the fundus of the gallbladder itself The concentrating mechanism of the gallbladder is intact and it empties normally after the fat meal

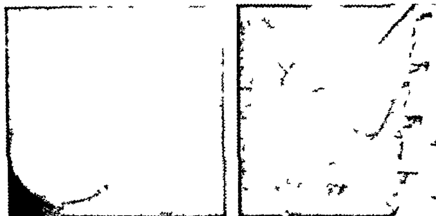


Fig 594 (*Left*) Congenital anomaly of the gallbladder (Phrygian cap)
Fig 595 (*Right*) Triloculation of the gallbladder

INTRAHEPATIC GALLBLADDER

The intrahepatic gallbladder in man is a developmental anomaly and is of not infrequent occurrence in lower animals such as the shark many reptiles the pig etc [1] It may be considered a result of arrested development since during the second month of embryonic life the gallbladder is buried in hepatic tissue becoming superficial at a later period [2]

LEFT SIDED GALLBLADDER

Schachner[3] collected 13 cases in the literature in which the gallbladder had been found on the left side A left sided gallbladder demonstrated roentgenologically was reported by Hartung[23]

GALLBLADDER ASSOCIATED WITH A MESENTERY

In some cases, the gallbladder is possessed of a 'mesentery' by which it hangs from the liver for a distance of 2 to 3 cm from the inferior hepatic surface This anomaly has been described in 4 to 5 per cent of the dissecting room specimens Such freedom of mobility may have considerable clinical importance because of the possibility of torsion on its pedicle with circulatory impairment Gross listed 50 cases of such a nature in his series

CONGENITAL ABSENCE OF THE GALLBLADDER

Mentzer[21] made a careful study of congenital anomalies of the bile ducts He reported the occurrence of 2 cases of congenital absence of the gallbladder in a series of 1 600 autopsies He collected 17 species of fish 9 species of birds, and 26 species of mammals which normally possess no gallbladder

Bower [4] described a case of his own of congenital absence of the gallbladder as noted at operation and reviewed in brief fashion all similar cases previously described in the literature

Absence of the gallbladder had been reported by Melville He collected 38 such cases described in the literature Dixon and Lichtman [6] in 1945 added 10 cases

In addition, cases of atresia of the bile passages have been described In contrast to this latter group there is no evidence of a rudimentary cord in the cases of congenital absence of the gallbladder This anomaly apparently arises because of the failure of the development of a gallbladder bud from the hepatic diverticulum

Illustrative Cases One of the most common congenital anomalies of the gallbladder is the Phrygian cap illustrated in Figure 594 As described in the text, this is the result of a septum of normal tissue invading the lumen of the gallbladder and producing a partial subdivision into an upper and a lower locule

A rare anomaly is triloculation of the gallbladder (Fig 595)



FIG 596 (A *Left*) Diverticulum of the gallbladder (B *Right*) Same patient as is shown in (A) Appearance after the fat meal

Another infrequent anomaly of the gall bladder is the presence of a diverticulum (Figs 596A and B) Figure 596A shows the appearance of the gallbladder when filled with the dye Figure 596B shows the appearance after partial emptying after the fat meal There is a concomitant diminution in the size of the diverticulum

Another rare anomaly of the gallbladder is the hook shaped type shown in Figure 597

The fact that a gallbladder exhibits a congenital anomaly does not of course preclude the presence of associated organic disease This is illustrated by the following case Roentgen examination in this case (Fig 598) revealed a gallbladder filled with the dye which had a septum almost completely dividing it into two fairly equal parts (Phrygian cap) A very faint mottling could be noted throughout but no clear cut sharply defined translucent areas At operation the presence of a septum consisting of normal tissue was found producing the biloculation seen in the roentgenogram In addition the gallbladder was filled with many small grape seed sized calculi

This is an example of a biloculation of the gallbladder of congenital origin due to a septum extending into the lumen of the organ In addition many small calculi were present



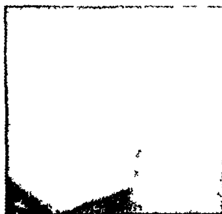


Fig 597 (*Left*) Hook shaped appearance of the gallbladder
Fig 598 (*Right*) Congenital anomaly of the gallbladder (Phrygian cap) with numerous calculi

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62

Biliary Fistulas

Internal biliary fistulas may result from the following

1 Primary disease of the gallbladder ulcerating through into the duodenum

2 Ulceration of the duodenum invading the gallbladder

3 Ulceration through the gallbladder invading the colon

4 A communication between the gallbladder and the stomach duodenum or jejunum may also be established as the result of operative interference

5 In rare cases fistulous communications have been found between the common bile duct and the duodenum and between the gallbladder and the stomach

6 In rare instances there may be a reflux of barium from the duodenum up through the ampulla of Vater outlining the biliary tree although no evidence of a direct fistulous communication can be found

The most common cause of a duodenobiliary fistula is the presence of primary gallbladder disease[1 9] Roentgenologically barium is noted escaping from the duodenum into the gallbladder and up into the biliary tree In many cases it may be impossible to determine the exact cause unless the calculus itself is demonstrable in the roentgenogram

The first roentgenologic demonstration of a cholecystoduodenal fistula the case having been checked by autopsy was that by Carman[10] in 1915 Busi[11] described a similar fistula in 1919 which he had observed on roentgenologic examination The roentgenogram however is not included in his written contribution

In Ludin s case[14] the marked dilatation of the small intestine was outlined by barium and within it was a negative shadow produced by a calculus These findings were controlled by autopsy and although a fistula was found between the gallbladder and duodenum this was not demonstrable in his roentgenogram

Procq Brodin and Aime[13] described a case of impaction of a large gallstone within the duodenal bulb which became visualized because of the barium which encompassed it the central zone being translucent The patient refused operation only to return at a later date for emergency surgical intervention because of an acute ileus A calculus was found obstructing the small intestine At autopsy a cholecystoduodenal fistula was found with another stone present at its mouth

In Crane s case not only was the fistulous communication between the duodenum and the gallbladder visualized but in addition the entire duodenal curve was dilated and terminated abruptly at the duodenojejunal junction where a large radiolucent biliary calculus was present The diagnosis was operatively confirmed Kaiser s experience[4] was of a somewhat similar character Roentgen examination showed a central translucent rounded area due to a biliary calculus occupying almost the entire lumen of the duodenal bulb The actual fistulous communication between the gallbladder and the duodenum was also demonstrable A later roentgenogram showed that the stone had escaped from the duodenal bulb and was present as a translucent zone in the jejuno ileal region where it produced partial obstruction The diagnosis was confirmed at operation The actual demonstration of the translucent shadow produced by the gallstone at the distal end of the obstructed and dilated jejunum the calculus

having escaped by way of the duodenum, is well illustrated in the case reported by Schele[6] In the cases described by Benassi[14] and Andersen,[15] the gallstone remained impacted within the duodenal bulb and produced a rounded translucent area outlined by a rim of barium

A cholecystoduodenal fistula may also be secondary to a primary carcinoma of the gallbladder A case of this nature in which the radiologic evidence of a fistula was confirmed at autopsy was reported by Beutel[16]

in one of the cases described by Lonnerblad[18] and is also described in the reports by Vorhaus[19] Startz[20] and Danzer[1] The roentgen diagnosis in such cases may be best made by means of the barium enema As the barium reaches the hepatic flexure, it may then escape into the gallbladder, completely filling it and, in addition, outlining the biliary ducts

A not uncommon cause of a communication between the gallbladder and the duodenum is cholecystoduodenostomy usually



FIG 599 Appearance following cholecystogastrostomy

McCorkle and Fong[1] described a case of cholecystogastric fistula due to a carcinoma of the stomach which had ulcerated into the gallbladder

A duodenal ulcer may also be the cause of a duodenobiliary fistula This was true in one of the cases reported by Lonnerblad[18] In the presence of a typical duodenal deformity one may assume that the fistula had resulted from the perforation of the ulcer into the biliary tract

An unusual type of fistula is that between the gallbladder and the colon This occurred

performed in cases of obstruction of the common duct When the gallbladder becomes filled as a result of the escape of barium from the duodenum the history of course gives a definite clue as to the etiologic factor The gallbladder may also be visualized as the result of an anastomosis between it and the jejunum

A rare type of fistulous communication between the duodenum and the common bile duct was described by Öhnell and Lindblom A fistula between the gallbladder and the stomach as the result of the



FIG 600 Barium and air in the bile ducts



FIG 601 Choledocho duodenal fistula

perforation of a calculus occurred in the cases reported by Graberger [3] and Pohlandt [4]

A retrograde filling of the biliary radicles with barium through the sphincter of Oddi in the absence of any fistulous communication was described by Beall and Jagoda and Wichtl [26] It is possible of course, that in such cases there may have been a very small fistulous communication between the

Such visualization of gas filled bile ducts is in itself definite proof of a fistulous communication between the biliary apparatus and a hollow viscus

Illustrative Cases The most common cause of a fistula between the stomach or the duodenum and the gallbladder is cholecystogastrostomy or cholecystoduodenostomy This is illustrated by the following case



FIG 602 (A *Left*) Choledocho duodenal fistula Note barium and air in the bile ducts (B *Right*) Same patient as is shown in (A) Note the common duct outlined by barium and the translucent areas at the obstructed distal end

gallbladder and the duodenum at the time of the original roentgen examination which later healed so as not to be recognizable at operation, or the opening may have been so small as to escape the eye of even a practised surgeon

Air in the bile ducts may occasionally be noted when an internal biliary fistula is present between the gallbladder and the duodenum and also between the gallbladder and the colon [27] The delineation of the bile ducts by air may be noted in the scout film prior to the administration of any barium

R D female aged 49 About 1 week before hospitalization this patient had an attack of severe pain in the epigastrium which radiated to both costal margins The pain recurred from then on and was intense

Physical examination revealed a globular mass palpable below the right costal margin It was exquisitely tender There was an icteric tint to the conjunctivae While on the ward the patient vomited once

Operation revealed that the gallbladder and biliary passages were quite markedly distended Arrangement of the bile ducts was abnormal in that the cystic duct did not join the hepatic duct to form the common duct





Fig 603 (A *Left*) Gallstone ileus Note (1) gas in the gallbladder (2) abnormal dilatation of the small intestine (3) biliary calculus (B *Right*) Same patient as is shown in (A) Appearance after injection of barium through the Miller Abbott tube Note the gas in the gallbladder and also the arrest of the barium by the calculus (C, *Center*) Same patient as is shown in (A) and (B) Roentgenogram of the calculus

until the upper level of the duodenum was reached The gallbladder wall was slightly thickened On the posterior surface of the duodenum at a point where the common duct entered it there was a round, hard, irregular mass about 1 inch in diameter which at first was thought to be an impacted stone at the extreme lower end of the common duct After

exploring the duct with a probe, and after a more thorough exposure however, it was thought that this tumor mass was due to a malignancy rather than to a stone A cholecystogastrostomy was performed The patient was discharged in good condition

Roentgen examination made after operation (Fig 599) showed the barium filled gall bladder to be attached to the greater curvature of the prepyloric portion of the stomach

The biliary tract may also become visualized by way of the duodenum, as is demonstrated by the next case

S Z male, aged 49 This patient had a 10-year history of epigastric pain it was most severe at night and was relieved by the ingestion of milk and soda He had lost 40 pounds in the preceding 3 years There was no vomiting or tarry stools The patient showed excellent improvement on an ulcer regimen

Roentgen examination revealed no definite evidence of any organic lesion of the stomach or duodenum Regurgitation of barium however, was noted filling up the common duct The intrahepatic ducts were visualized because of the presence of air within them (Fig 600)

As the patient made an excellent clinical recovery on medical management surgical intervention was not attempted The most likely explanation is that at the time of roentgen examination a fistula had developed between an ulcer of the duodenum and the common bile duct even though the ulcer itself could not be definitely shown Not only the roentgen evidence of barium and air in the bile ducts but also the excellent clinical response to ulcer therapy may be explained on this basis A less likely explanation is that a spontaneous regurgitation of barium took place by way of a relaxed sphincter of Oddi



Fig 604 Cholecystocolic fistula

In the next case duodenal ulcer as the cause of the biliary fistula was checked by surgical intervention

R Z male aged 49 The patient gave a 2½ year history of recurrent attacks of epigastric pain relieved by food He vomited at times the vomitus occasionally containing coffee ground material He had one severe hemorrhage with tarry stools, necessitating transfusion He had lost 25 pounds in the preceding 6 months

Physical examination of the abdomen was essentially negative

Operation disclosed a duodenal ulcer and a fistula between the duodenum and the common bile duct where the ulcer had perforated into the duct

Roentgen examination (Fig *601) revealed*

a deformity of duodenal ulcer and the entire common duct which was outlined by barium which had passed into it because of the fistulous communication between the ulcer and the common duct

The following is an example of a choledochoduodenal fistula the result of an anastomosis which was made because of common duct obstruction Barium on reaching the duodenum escaped into the common duct and then by way of the hepatic duct into some of the smaller biliary radicles (Fig 602A) Air is also present in the intrahepatic bile ducts With less barium in the pyloro-duodenal region, the entire course of the common duct may be visualized (Fig 602B) It is moderately dilated There is a cup shaped defect at its distal end and a small round translucent area proximally evidently due to calculi

A calculus escaping into the intestinal tract from the gallbladder may cause intestinal obstruction

A K female aged 75 The patient was essentially well until 5 days before admission when she developed colicky abdominal pain constipation and fecal vomiting Physical examination revealed abdominal distention and tenderness Operation revealed a large biliary stone in the ileum 2 feet from the cecum and two small stones proximal to the large one Adhesions very dense in nature were found in the region of the gallbladder and proximal small bowel

Roentgen examination (Fig 603A) showed three important features (1) air outlining the gallbladder (2) abnormally dilated loops of small intestine and (3) evidence of a large biliary calculus The diagnosis of acute obstruction of the small intestine by a gallstone was obvious on the basis of the roentgenologic evidence A small amount of a suspension of barium injected through the Miller Abbott tube further emphasized the obstruction produced by the calculus (Fig 603B) Even if the calculus had not been demonstrable the air in the gallbladder would have been the clue to indicate that a fistula existed which permitted the escape of a stone into the intestine where it became impacted Fig 603C shows the appearance of the calculus removed at operation

The following case illustrates a fistulous communication between the hepatic flexure of the colon and the gallbladder

A B female aged 55 Ten months before admission to the hospital the patient developed diarrhea with four to six bowel movements a day The stools were soft but never watery No blood was noted in them She experienced no cramps pains vomiting chills or fever She believed that she had lost considerable weight Physical examination was essentially negative

From the history a disturbance of the large intestine was suspected and an examination of the colon was made by means of a barium enema When the barium reached the hepatic flexure some escaped into the gallbladder and cystic duct The roentgenogram showed a normal hepatic flexure Just above it was the gallbladder filled with barium The cystic duct was visualized and was outlined partially by barium and partially by gas within it (Fig 604)

The diagnosis was cholecystocolic fistula The patient refused operation

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63

Cholangiography

An important chapter in the history of roentgenology of the gallbladder and bile ducts was introduced with the development of cholangiography. In this procedure the gallbladder and bile ducts are visualized after the direct introduction of an opaque substance. The opaque substance may be injected through a biliary fistula by way of a T tube inserted for postoperative biliary drainage or directly into the gallbladder at the time of operation. Tenney and Patterson[1] were among the first to visualize the bile ducts roentgenologically by direct injection of a bismuth paste through an external biliary fistula. The roentgenogram showed the intrahepatic ducts as a result of an obstruction at the junction of the hepatic and the common ducts. Reoperation disclosed the fact that the common duct was practically obliterated by adhesions. Similar attempts to visualize the biliary tract by injecting a postoperative biliary fistula were made by Carnot and Blamoutier, who employed a suspension of barium; Cotte,[3] who injected lipiodol; and Walzel,[4] who introduced 15 to 20 cc. of iodipin.

Of considerable value is the direct introduction of an opaque medium through the T tube. Various media may be employed, such as lipiodol, umbrathor, thorotrast, hippuran or diodrast. I have found diodrast to be superior to iodized oils as a medium in cholangiographic visualization of the biliary tree. Because of its more fluid state it tends to fill out the bile ducts in a more homogeneous manner. The iodized oils are apt to show a separation into discrete globules even when introduced at body temperature. Because of this some of the ducts may show small rounded discrete areas of opacity, which introduces an element of difficulty in determining with assurance whether any of the intervening parts of the duct not completely outlined by the opaque oil are actually normal. There is also a freer flow of the diodrast through the common duct into the duodenum. With less lag a small calculus is not as readily obscured as it may be by an overaccumulation of the thicker oily medium. Although the diodrast flows very rapidly into the duodenum, it need not interfere with the completely satisfactory visualization of the common duct as well as the intrahepatic biliary radicles.

The following is the procedure which I have employed. Conditions should be as aseptic as possible. The diodrast is first warmed to approximately body temperature by placing the vial in a basin containing warm water. 20 cc. are then drawn up into the sterile syringe and all the air expelled. When there is a free flow of bile from the rubber tube it may be clamped while the bile is escaping and any air within the tube will therefore have been eliminated. The tip of the syringe is then inserted into the end of the rubber tube and held vertically so that air which may not have been entirely expelled will rise to the surface. A small amount of bile may be withdrawn if desired before the diodrast is introduced. The injection is first done under fluoroscopic control, just enough being introduced to make sure that the T tube is in place and that the common duct is visualized. The tube is then clamped off. The cassette is set in place with the tube centered over the biliary tract area of the patient, who remains in the supine position, and the roentgen exposure technic is set for immediate exposure at the desired moment.

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an added reason for avoiding overdistention of the pancreatic duct when possible

The cholangiogram may show evidence of calculi in the common duct which had been previously overlooked at the time of cholecystectomy Such findings are well illustrated by a number of reports

A calculus within the common duct may exhibit the following evidence of its presence in the cholangiogram

1 It may appear as a rounded translucent area within the confines of the duct completely surrounded by contrast substance

2 When of large size producing occlusion the calculus may produce a cup-shaped defect at the distal end of the duct

3 If the calculus is closely adherent to one wall it may appear as a sharply outlined defect in the contour of the duct

4 At times one may have the combination of an irregularly outlined calculus and close adherence to the wall in which event the area of involvement may appear irregularly narrowed as if by an infiltrating lesion The injection of additional contrast substance may aid materially in ultimately showing its intraluminal position and the freedom of invasion of the wall itself

5 A calculus particularly if small may be completely obscured when the duct is filled with contrast substance

This diagnostic error may be avoided in the following ways

1 By the fractional method of examination of injecting only 5 cc of contrast substance before each roentgen examination In this way a calculus may be diagnosed in the first roentgenogram which may not be seen when the duct is more completely filled

2 A further aid is in the roentgen observations made during the course of the emptying of the duct into the duodenum As the quantity of the contrast material diminishes a hitherto obscured translucent area may come into view

In addition to the recognition of calculi strictures of the common duct may be recognized by the failure of the opaque material to pass through the involved region The constricted region itself may be noted when the narrowing is only partial with secondary dilatation of the proximal portion of the biliary tree When the stenosis is complete only the distended bile radicles will be demonstrable with none of the opaque medium outlining the distal end of the duct

In obstruction of the sphincter by a tumor of the head of the pancreas the cholangiogram may show an abrupt termination of the distended common duct sometimes associated with irregularity of the contour at that site

Not only may obstruction of the common duct result from organic disease but it may also be secondary to disturbances of a functional nature as the result of biliary dyskinesia In such cases the entire common duct as well as the biliary tree is readily observed because of spasm at the sphincter The common duct tapers in a smooth manner and there is no evidence of any organic disease Following the administration of 1/100 grain of glyceryl trinitrate or after the inhalation of amyl nitrite there will be complete relaxation of the sphincter permitting the opaque material in the bile radicles and in the common duct to make its ready escape into the duodenum On the other hand the hypodermic administration of ¼ grain of morphine exaggerates the spasm of the sphincter of the common duct If a cholangiogram is obtained following this procedure evidence of spastic obstruction at the sphincter will be indicated by the distention of the common duct and the visualization of the intrahepatic biliary radicles as well as by the prolonged delay in the emptying time The effectiveness of the administration of nitroglycerin in alleviating spasm at the sphincter of the common duct is well shown in one of the cases reported by Hunt Hicken and Best in which a closure of the distal end of the common duct continued for 14 days after operation as demonstrated by cholangiography Relief

The clamp is removed and 5 cc of the fluid are introduced slowly At the moment of completion the tube is clamped, the patient is instructed to stop breathing and an immediate roentgen exposure is made This process is then repeated, following the introduction of an additional 5 cc of the fluid, until four such successive exposures have been made (a total of 20 cc having been injected in the average case) During this procedure the introduction of the fluid must be done gently and the rate slowed when necessary if resistance is met with or if the patient complains of right upper quadrant discomfort *This is usually an indication that the intrahepatic ducts are being distended too rapidly*

After the fourth exposure is completed the tube is clamped and an additional roentgen examination is made in the right lateral position The patient remains on the table with the tube clamped and another roentgenogram is taken 15 minutes later in order to determine the emptying of the biliary tract The films are all developed promptly If the 15 minute film shows satisfactory elimination of the opaque fluid the examination is completed Depending on the degree of delay additional films may be taken at 15 minute or ½ hour intervals *until the patency of the biliary tract is determined*

The advantage of injecting only 5 cc of the diodrast at a time is that it enables one to visualize the biliary tract with varying degrees of distention A tiny calculus may be seen through a small amount of the diodrast which may otherwise be obscured when the ducts are overdistended On the other hand, if only a moderate amount is injected the entire extent of the biliary tree may not be visualized because not enough material is present to reach every part of it In the *event that the injection* meets with considerable resistance when the larger amount is added either the rate may be slowed still further or if necessary the procedure may be terminated By making the exposures promptly after each injection the appearance of the biliary tract at the moment will be fixed in the roentgenogram before the opaque fluid has had an opportunity of escaping into the duodenum, particularly when the sphincter offers little or no resistance The value of the right lateral roentgenogram is that occasionally it makes possible a clearer visualization of the isolated common duct

This *serial cholangiography* obviates the necessity of the preliminary administration of a hypodermic of morphine in order to constrict the sphincter as an aid in the distention of the biliary tree Following the *method which I have outlined, I have never* experienced any difficulty in the visualization of the entire length of the common duct *and of the intrahepatic ducts* More over there are in my opinion a number of objections to the preliminary hypodermic injection of morphine First there may be abnormal distention of the duct with the opaque medium, so that a small calculus may not so readily show as an intraluminal translucent area Secondly it may interfere with a study of the rapidity with which the common duct empties under normal conditions

A third objection to the abnormal spasm of the sphincter produced by the morphine *is that it may force the opaque medium* more readily into the pancreatic duct Even in the absence of morphine injection the pancreatic duct is occasionally visualized While the demonstration of the pancreatic duct is theoretically interesting it is questionable whether a definite effort to fill it with the opaque medium is advisable Certainly there would be an objection if there were any doubt as to the sterility of the biliary tract

In this connection, the observations of Liedberg[5] are of interest In 8 out of 50 cases in which a reflux of opaque medium had occurred into the pancreatic duct, he found a pathologic diastasuria suggesting that some degree of injury to the pancreas may occasionally take place While the evidence is only suggestive it is certainly

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was obtained 10 minutes after the administration of 1/100 grain of nitroglycerin placed under the tongue

Cholangiography is, therefore, of considerable diagnostic value in the demonstration of organic as well as functional disturbances and the findings give a definite clue as to the therapeutic approach Routine cholangiography adds considerable certainty as to the advisability of removal of the T tube

Occasionally the pancreatic duct may be visualized during the course of cholangiography [6 7 8 9] In some of these cases a calculus at the ampulla of Vater was found which conceivably, by obstruction, was a factor in permitting the delineation of the pancreatic duct The fact that the pancreatic duct can be visualized during cholangiography is one reason among others why aseptic conditions are essential during this procedure



FIG 605 Normal cholangiogram The gallbladder the cystic duct, the intrahepatic ducts, the common duct and the distal end of the pancreatic duct may be seen The duodenum is also visualized by the opaque fluid which has entered it

Cholangiography may also be attempted during the course of operative intervention [10]

The procedure for operative cholangiography is *effectively* carried out in the following way

1 A 14 by 17 cassette with a stationary grid over it is put into a wooden tunnel which has been placed on the operating

table under the patient so as to include the upper portion of the abdomen

2 A No 20 needle attached to a piece of rubber tubing about 20 centimeters long is inserted directly into the common duct

means of a portable x ray machine is made simultaneously with the completion of the injection This process is repeated and another roentgen exposure is made after the injection of an additional 10 cc of the fluid



Fig 606 Normal cholangiogram

3 A 50 cc syringe is filled with the contrast substance and attached to the rubber tube A small amount of bile is slowly aspirated and with respiratory movements of the patient momentarily arrested by the anesthetist about 10 cc of the contrast substance is injected Roentgen exposure by

The films are developed immediately Additional exposures may be made with and without the introduction of more of the contrast fluid

An important practical application of cholangiography was emphasized by Walters and Weson[11] After employing cholan



Fig 607 (A, *Left*) Normal cholangiogram Note the obliteration of the distal end of the common duct (B, *Right*) Same patient as is shown in (A) One minute later showing the distal end of the common duct open

Fig 608 (A, *Left*) Note indentation at the medial border of the common duct at its distal end as well as the pancreatic duct (B *Right*) Same patient as is shown in (A) *About 1 minute later Note the change in appearance of the distal end of the* common duct The opaque material is now in the duodenum

giography for the visualization of a common duct stone they instilled ether into the *common bile* duct which caused fragmentation of the stone In addition inhalation of amyl nitrite caused dilatation of the sphincter of Oddi thereby aiding in the expulsion of the fragmented calculus

A normal cholangiogram is illustrated in Figure 605 Diodrast was injected through a rubber tube inserted into a draining biliary fistula The biliary tree is well visualized The common duct is of normal lumen and smooth contour tapering at its distal end It empties readily into the duodenum

Illustrative Cases In the following case the normal cholangiographic findings were corroborated by autopsy

F K female aged 54 Five months before her admission to the hospital the patient had a severe attack of epigastric pain associated with jaundice nausea and vomiting lasting for 2 weeks One month before her admission the jaundice recurred again associated with epigastric distress nausea vomiting and clay colored stools The patient had noticed blood in the stool during the preceding 3 weeks She had lost 21 pounds in the month before admission Physical examination revealed marked jaundice of the skin and sclerae Examination of the abdomen was essentially negative except for tenderness in the right upper quadrant

Exploratory laparotomy revealed the stomach duodenum liver and bile ducts to be normal The head of the pancreas was somewhat hardened and slightly enlarged and thought to be inflammatory in nature The common bile duct was opened and a T tube was inserted Her symptoms subsided

Cholangiographic examination (Fig 606) revealed the gallbladder cystic duct biliary tree and common duct to be normal Three months later there was a recurrence of the jaundice with mild crampy epigastric pain and clay colored stools The icterus cleared up in about 5 weeks

Four months later she was readmitted with persistent jaundice pain mainly in the right upper quadrant progressive fatigue and occasional clay colored stools The abdomen had become markedly distended

Physical examination of the abdomen at this time showed the presence of fluid Abdominal paracentesis resulted in the drainage of at least 3 000 cc of yellowish clear colored fluid This was repeated with a similar result 11 days later Examination of the ascitic fluid showed no tumor cells

Autopsy revealed a toxic cirrhosis of the liver with ruptured varices of the esophageal veins There was no abnormality of the gall bladder or bile ducts

The functional behavior of the sphincter



FIG 609 Visualization of the pancreatic duct

with its ability to constrict and dilate is well illustrated by the following case

A B female aged 55 The patient had had a cholecystectomy for chronic cholecystitis with cholelithiasis and a common duct exploration with insertion of a T tube

The patient made an uneventful recovery

Cholangiography after the injection of diodrast showed complete obliteration of the dis

tal end of the common duct (Fig 607A) The rest of the biliary tree was normal Figure 607B shows the appearance about 1 minute later The distal end of the common duct is now fairly widely open The common duct is entirely normal in appearance There is no dilatation and there are *no intraluminal* translucencies

Changes in the contour of the common having emptied its content into the duodenum

The visualization of the *pancreatic duct* during cholangiography is illustrated by Figures 609 and 610

The most valuable application of the cholangiogram is in the demonstration of calculi within the ducts A stone in the com



Fig 610 Visualization of the entire length of the pancreatic duct

duct during emptying may be noted in Figures 608A and B In Figure 608A there is an indentation of the medial border of the common duct The pancreatic duct is visible In Figure 608B, about 1 minute later the contour of the common duct has changed The extreme distal end is now visible tapering smoothly as it enters the duodenum which now contains diodrast The pancreatic duct is no longer visible presumably

mon duct may produce obstruction and prevent the ready flow of the contrast substance into the duodenum The defect at the distal end of the duct will vary with the degree of obstruction and the shape of the stone The deformity will also show considerable alteration in the same individual in observations made during the course of the examination as more of the diodrast encompasses the stone Figure 611 shows a

translucent area at the distal end of the common duct In addition there is an ex-



FIG 611 Calculi at the distal end of the common duct Only the translucent area produced by the large calculus is clearly seen

tremely small rounded translucent area proximal to this On reoperation several calculi were removed from the common duct

When the stone is very large it may completely block the common duct and produce a cup shaped defect

L D male aged 56 One year before examination the patient had a severe attack of pain in the epigastrium which radiated to the right side and was associated with fever but no vomiting He remained well for 11 months when he had recurrences of severe upper abdominal pain With the last attack he vomited bile colored fluid He was intensely jaundiced and soon thereafter while hospitalized became semicomatose He was operated on promptly and a cholecystostomy was done No stones were present in the gall bladder The common duct was not explored The patient made a prompt recovery His jaundice cleared and several weeks later a cholangiographic examination was made by injection of diodrast into the tube (Fig 612A) This disclosed considerable dilatation of the common duct and a cup shaped defect at the distal end The diagnosis of an impacted calculus in the common duct was made At the second operation the stone was successfully removed (Fig 612B)

A cup shaped deformity produced by a calculus is illustrated by the following case



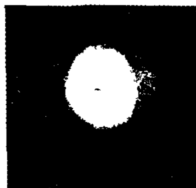


FIG 612 (A *Left*) Obstruction at the distal end of the common duct by a calculus (B *Right*) Same patient as is shown in (A) Appearance of the gallstone removed at operation



FIG 613 Calculi in the common duct



FIG 614 (A *Left*) Irregularity in the common duct produced by caculi (B *Right*) Same patient as is shown in (A) Appearance on re examination showing intraluminal position of the calculi

T G female aged 59 The patient was operated on at another hospital where a cholecystostomy was done A tube was inserted into the gallbladder On her admission to this hospital a cholangiographic examination was made (Fig 613) A cup-shaped defect was noted near the distal end of the dilated common duct There was another vaguely outlined translucent area about 1 inch proximal to the defect just described The gallbladder and cystic duct were outlined

G O female aged 64 During the preceding 4 or 5 years the patient had recurring attacks of nonradiating pain in the right upper quadrant She was jaundiced on one occasion and had an intolerance to fatty foods Physical examination of the abdomen was essentially negative except for tenderness in the right upper quadrant The pain at times was severe at night

Operation revealed the gallbladder to be considerably enlarged The common duct was



Fig 615 Stricture of the common duct Note the overdistention of the biliary tree proximal to the zone of constriction

The report at operation was as follows The common duct was found to be dilated and two stones shown in the cholangiogram were palpable The gallbladder was diseased

Both stones were removed A cholecystectomy and choledochostomy were done with T tube drainage Cholangiography after operation showed the common duct to be free of any intraluminal translucencies

Various abnormalities produced by calculi in the common duct may be noted in the next case

tremendously dilated and contained two stones A cholecystectomy was done The common duct was opened the two stones were removed and a T tube inserted

Cholangiographic examination after operation (Fig 614A) revealed the following findings

1 Considerable dilatation of the common duct and obstruction at its distal end

2 Translucent areas within the common duct and considerable irregularity in the outline of its distal portion This was apparently due to the fact that the diodrast at this particular moment had not been able to filter



Fig 616 Stricture of the proximal portion of the common duct

through between the calculus and the wall to which it was closely adherent

Re examination (Fig 614B) showed the distal translucent area to be intraluminal in position the contrast fluid having found its way between the wall of the duct and the calculus itself

The diagnosis then on the basis of the cholangiographic findings was calculi within the common duct

The report at autopsy was 'Upon opening the second portion of the duodenum the papilla of Vater is prominent The sphincter of Oddi is tightly closed and a hard mass can be felt in this region When the common duct is open from above two stones of conical shape are found completely obstructing the lumen Each measures about 1 cm in diameter

The cholangiogram may be useful in the demonstration of a stricture of the common duct

E C male aged 27 The patient had had a cholecystectomy for cholelithiasis Drainage subsided after 2 months and he remained well for 1 month thereafter From then on he continued to have attacks of severe pain in the right upper quadrant at times requiring a hypodermic of morphine and frequently associated with fever (as high as 104 F), jaundice and clay colored stools He lost 17 pounds

On physical examination there was tenderness in the right upper quadrant The operative scar was well healed The preoperative clinical diagnosis was stone in the common duct The possibility of a stricture of the common duct was also considered

Operation revealed a stricture of the common duct There were no calculi The common duct was drained

Figure 615 shows the appearance after the introduction of diodrast In the midportion of the common duct there is a narrowed area proximal to which the duct is dilated Distal to this narrowed zone the common duct balloons out again and is normal in appearance

Postoperative kinking of the common duct is illustrated by the next case

G M female aged 31 The patient had had recurring attacks of colicky epigastric pain radiating to the back and associated with vomiting Roentgen examination of the gall bladder showed numerous calculi A cholecys



Fig 617 Fistula of the common duct

tectomy and appendectomy were done The gallbladder was inflamed and filled with stones The appendix was normal Several days after operation she became jaundiced and had fever her temperature rising to 103 6 at the drawn toward the gallbladder bed at a point where the cystic duct joined the common hepatic duct This was stenosed and the cystic duct itself contained inspissated bile Once the common duct was opened, liver bile



Fig 618 Fistula of the common duct Note the distention of the intra hepatic radicles and the absence of any of the contrast substance in the distal portion of the common duct The rubber tube introduced into the fistula is well outlined the radiographic exposure having been made during the course of the injection of the opaque fluid

end of 10 days She then developed a fistula through which the bile drained externally The patient was readmitted 2 months later because of recurrent attacks of colicky right upper quadrant pain radiating to the back and associated with chills and moderate jaundice

Operation at this time revealed that the common bile duct was kinked and apparently escaped from the upper portion of it and a probe introduced into this opening passed for a distance of 3 inches into the liver substance without encountering any stones

Similarly a probe introduced in the lower portion of the common duct passed for a distance of 2½ inches without encountering stones After the T tube was inserted into

these two portions of the duct 60 cc of injected saline apparently entered the duodenum and there was no backflow about the tube"

Roentgen examination (Fig 616) after injection of the contrast substance (in this case lipiodol) showed the strictured and kinked area in the common duct just distal to its junction with the cystic duct Lipiodol could be forced through this narrowed region The rest of the common duct was of normal contour Its lumen was diminished but it emptied readily into the duodenum

The intrahepatic ducts were markedly distended, due to the obstruction produced by the kink in the common duct Although diodrast is preferred in cholangiography lipiodol as already stated was used in this case Note the tendency to fragmentation within the intrahepatic ducts an appearance which is never noted after injection of diodrast



Fig 619 (A *Left*) Obstruction of the common duct by a tumor of the pancreas Note the smooth abrupt termination of the dilated common duct and the distended intrahepatic radicles (B *Right*) Same patient as is shown in (A) Appearance in the right lateral position

Cholangiographic examination may show the location of a biliary fistula

J T female, aged 22 The night before her admission to the hospital the patient developed severe epigastric pain radiating to the back and associated with nausea and vomiting She had had an appendectomy 13 years previously and 2 years thereafter was operated on for adhesions From then on she had recurring attacks of pain similar to that which brought her to the hospital Cholecystography at one time showed a failure of visualization of the gallbladder shadow

Physical examination revealed no evidence of jaundice She was extremely tender in the right subcostal region There was moderate rigidity and marked rebound tenderness over the entire abdomen

At operation, the pancreas felt indurated The gallbladder appeared normal but contained hundreds of small brownish faceted calculi, of which there were three in the cystic duct A stone was also removed from the common duct A cholecystectomy was done and a T tube inserted into the common duct The patient continued to drain bile profusely through the T tube The stools were acholic indicating that none of the bile was reaching the duodenum On reoperation there was a fistulous tract which could be traced to the point where the T tube had been inserted In this area there was a complete inflammatory obliteration of the middle third of the common duct The pancreas and remaining common duct were normal The flexible probe inserted into the sealed-off distal end of the common duct passed easily into the duodenum No stones could be felt in the hepatic ducts or in the first portion of the common duct The common duct was reconstructed the original T tube having been previously removed

Roentgen examination (Fig 617) showed

the biliary tree and the common duct filled with the opaque material The fistula was in the common duct through which the tube for injecting the opaque material was inserted None of the opaque substance was noted in the duodenum nor had any appeared in the intestine at the end of 30 minutes

The diagnosis was fistula of the common duct apparently associated with an obstructive lesion at the distal end of the duct

As noted at operation the fistula in the common duct was associated with a complete stricture of the duct just distal to it

The intrahepatic biliary radicles are visualized in Figure 618 after injection of the contrast substance into a biliary fistula associated with obstruction of the common duct

Obstruction of the common duct may result from a tumor of the pancreas

N S male aged 56 Fifteen months before his admission to the hospital the patient became suddenly jaundiced and had associated itching of the skin The stools were clay colored There was no abdominal pain The jaundice was progressive At operation a cholecystostomy was performed A firm mass in the region of the head of the pancreas was palpated The surgeon was not certain whether the lesion was due to a carcinoma of the head of the pancreas or to a pancreatitis During the next 5 months the patient continued to drain 20 to 30 ounces of bile every day through the cholecystostomy tube and in the interim had lost about 30 pounds in weight A reoperation was performed After dense adhesions were separated the pancreas was found to be uniformly enlarged and firm and the base of the gallbladder was markedly thickened thus preventing any operative procedure Whether neoplasm of the pancreas or just chronic inflammation was present could not be determined

Examination of the biliary tree after the injection of the opaque material (Fig 619A) revealed the common duct completely outlined considerably dilated and sharply delimited at its distal end None of the contrast substance was noted entering the small bowel There were no negative shadows within the common duct The arborization of the intrahepatic biliary ducts was well shown Examination in the right lateral position (Fig 619B) showed essentially similar findings

The final diagnosis was obstruction of organic origin at the terminal end of the common duct This type of obstruction may be due to a tumor of the head of the pancreas

As noted in the findings at the two laparotomies the cause of the obstruction was an enlargement of the head of the pancreas the histologic nature of which was not determined because no biopsy specimen was taken

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THE SPLEEN, LIVER AND PANCREAS

64

Roentgen Visualization of the Spleen and Liver

THE SPLEEN

The spleen may occasionally be seen in the preliminary film particularly when the exposure is made with the aid of the Bucky diaphragm Such visualization is occasionally aided when the colon in the region of the spleen contains considerable gas or when the colon is artificially distended through the rectal injection of air

A tumor of the spleen when large may be recognized by the displacement which it produces In an upward direction it may elevate the left diaphragm The stomach may be displaced to the right and the left half of the transverse colon as well as the splenic flexure may be displaced in a downward direction The kidney and ureter as observed in the pyelogram may exhibit various degrees of dislocation In the posteroanterior view however the identification of the mass as being necessarily of splenic origin may be difficult These findings may also be produced by a tumor in the tail of the pancreas by a retroperitoneal sarcoma or by a tumor of renal or suprarenal origin Examination in the left lateral erect position may disclose the fact that the pressure defect is on the anterior wall of the stomach and that it is not retrogastric thus helping to eliminate a mass of retroperitoneal origin as the cause

A further aid of considerable diagnostic value is the demonstration of esophageal varices While the occurrence of such varices and a mass in the left upper quadrant might conceivably be a mere matter of chance the probability however, would be very strong that the tumor was of splenic origin as in Banti s disease

Calcifications in the spleen are most frequently due to tuberculous foci and to phleboliths Calcerous areas in the spleen may also be due to calcification of the larvae of *Pentastoma* Such findings may also be present in the liver Roentgen evidence of calcification in the spleen produced in this manner has been reported by Saupe[1] and Bignami and Secomandi As a rule differential diagnosis between calcifications in the spleen due to tuberculosis phleboliths or parasites on the basis of the roentgen appearance is often difficult or impossible

The view that these miliary areas of calcification frequently represent healed tuberculous foci is supported by the following observations

1 There is morphologic similarity between some of these lesions and healed tuberculous foci located elsewhere (Mitchell[3] Moorman[4] and Reichle and Work[5])

Sweany[6] showed the histologic similarity of the splenic tubercles to those occurring in the lungs He also emphasized the relative frequency of such calcifications of the

spleen in tuberculous patients examined at postmortem

2 In most of these cases one may demonstrate an active or an old tuberculosis in the individual, usually in the lung

3 Evidence indicates that some of these lesions harbor the tubercle bacillus Shands[7] implanted tuberculosis in guinea pigs by innoculation with material obtained from such lesions

4 Next to the lymphatic nodes, the spleen is the abdominal organ most often affected by tuberculosis This was shown by Winternitz[8]



Fig 620 Calcification of the splenic artery

There appears to be little doubt therefore, that some of these tubercles represent calcified tuberculous lesions That tuberculosis, however is not the only cause is shown by the well controlled microscopic studies of Barsony and Schutz[9] who showed that in their cases the calcifications were due to phleboliths Similarly Berman[10] showed by histologic study in three cases in which miliary calcifications of the spleen were noted roentgenologically that they were due to phleboliths although two of these cases showed primary tuberculosis of the lungs The lesions in the spleen were decalcified and examined microscopically The appearance was that of an organized calcified thrombus with an atrophying vein wall

Calcification of the splenic artery may show as a tortuous tubular structure in the anatomical location of this vessel The findings in Heatley s case[11] were controlled by operation The roentgen evidence described by Israelski[1] was subjected to confirmation at autopsy

A more significant type of calcification is that of aneurysm of the splenic artery This is exemplified in the completely proved cases of Lindboe,[13] Sperling[14] and Seids and Hauser[15] Roentgenologically, the lesion appears as a spherical mass with a calcific rim in the anatomic region of the splenic artery It may mimic the appearance of a calcified cyst of the spleen or of the tail of the pancreas or when close to the vertebral column it may resemble an aneurysm of the abdominal aorta Clinical aid in the recognition of the aneurysmal nature of the lesion may be obtained by the demonstration of pulsation of the mass on palpation and a bruit on auscultation Such findings of course will not necessarily prove that the aneurysm has arisen from the splenic artery unless it corresponds to the position of this vessel and is sufficiently far removed to the left of the aorta

Calcification of the spleen may also occasionally be secondary to intracapsular hemorrhage[16]

Calcification of an infarction of the spleen may be recognized roentgenologically by the tendency of this lesion to assume a triangular shape with the apex at the center and the base at the capsule This area is irregularly calcific in appearance but quite sharply delimited[17] In one of the cases reported by Kadrnka and Babaiantz there were present in the spleen two opposing triangular areas of calcification connected with each other The findings were controlled by anatomic proof with identification of the lesion in the roentgenogram of the removed spleen

A case of calcified perisplenitis with evidence of calcification of the splenic capsule has been reported by Stott and Cotton Cornwall[18]

CYST OF THE SPLEEN

The comparative rarity of cyst of the spleen is indicated by the fact that the autopsy records at Bellevue Hospital from 1904 to 1940 disclosed only 8 cases during which period there were 48 cysts of the liver[19]

This rarity is also shown by the fact that Fowler [?] in 1940 found only 137 cases in the literature from the time of the original report by Andral in 1829 An additional 11 cases from the literature were included in the contribution by McClure and Altemeier[21]

Occasionally a cyst of the spleen may be recognized by calcification of the wall (Gartesleben[2])

The calcified cyst of the spleen reported by Baumann and Kohnstamm[23] in 1930 is illustrated only by a drawing of the roentgen findings The autopsy report however is interesting in that the cyst of the spleen was really the result of an aneurysm of the splenic artery

The roentgen appearance of calcification of a cyst of the spleen is further exemplified by Shawan [4] Culver Becker and Koenig [5] Elkeles and James [6] and Neidhardt [7] Rarely a calcified cyst of the spleen may be multiloculated[23]

When the wall of a tumor occupying the left upper quadrant is calcified one may safely assume that the mass represents a cyst of the spleen particularly if it is associated with the characteristic displacements produced by a splenic tumor as already described



FIG 621 Enlargement of the spleen (Banti s disease) producing downward displacement of the left half of the transverse colon

Figure 620 shows the appearance of calcification *of the splenic artery* Note also the calcified splenic artery shown in Figure 629A

Illustrative Cases The spleen when enlarged may displace the left half of the transverse colon in a downward direction

F S P male aged 63 For 1 year the patient had been complaining of dyspnea and abdominal distention Physical examination of the abdomen revealed a firm mass in the left upper quadrant extending downward to the umbilicus There was evidence of fluid in the abdomen There were no telangiectasia

The liver edge was not palpable Following paracentesis 2 800 cc of clear light yellow fluid were removed The blood Wassermann was 4 plus A hypochromic anemia was also present

Given a mass in the left upper quadrant the origin of which is in doubt, evidence of esophageal varices would justify the conclusion that the tumor was of splenic origin As noted at operation and in the report of the pathologic examination the mass was a huge spleen the histologic appearance of which was consistent with the diagnosis of Banti s disease

The enlarged spleen may cause pressure deformities of the greater curvature of the stomach



Fig 622 Pressure deformity of the greater curvature of the stomach by an enlarged spleen

At operation there were 600 cc of clear straw colored fluid in the peritoneal cavity The liver was slightly enlarged The surface was smooth The spleen was the size of a foot ball A splenectomy was done and a biopsy of the liver was taken

The pathologic diagnosis was (1) Fibrosis of the spleen the histologic changes were compatible with those seen in Banti s syndrome (2) portal cirrhosis

Roentgen examination (Fig 621) revealed marked downward displacement of the left half of the transverse colon Examination of the stomach had also shown considerable displacement to the right Evidence of varices was found in the examination of the esophagus

M D, female aged 46 Seven months before her admission to the hospital she had complained of anorexia weakness dyspnea on exertion and a few months later of a productive cough without blood Soon thereafter she complained of upper abdominal crampy pain occurring from 15 to 30 minutes after meals occasionally radiating into the chest and down the arms and relieved on lying down She lost 36 pounds in the preceding year While on the ward she ran a low grade fever It was the opinion of the chest consultants that the patient had a chronic suppurative pulmonary lesion of undetermined etiology Physical examination of the abdomen was essentially negative

At operation the liver was found to be en

larged The spleen was enlarged and nodular, and a splenectomy was done

The report of the microscopic examination was The spleen is greatly enlarged weighs 510 gm and measures 17 by 11 cm It was received unfixed It has the normal configuration but no notches are present The surface has many protruding coarse nodules which elevate the bluish red smooth capsule It is firm but pliable and cuts without increased resistance About 90 per cent of the pulp is replaced by partly confluent masses of pinkish gray viable tissue resembling tumor tissue Between these masses small sunken areas of dark red splenic pulp persist The growth is quite uniform throughout the organ

The diagnosis after microscopic examination was granulomatous lesion of the spleen of undetermined etiology probably tuberculous

Roentgen examination (Fig 622) showed a pressure defect of the greater curvature of the pars cardia of the stomach The mucosal structure of the stomach in this region was intact and for this reason it was believed that there was an extra gastric mass in the left upper quadrant compressing the greater curvature of the stomach

A cyst of the spleen may not only displace neighboring viscera but may also show calcification of its contour

B S male aged 64 Nine months before his admission to the hospital the patient had complained of a gnawing epigastric pain made worse by food and somewhat relieved by alkalies and vomiting There was no blood in the vomitus or stools Physical examination of the abdomen revealed the liver edge to be four fingers breadth below the right costal margin firm smooth and not tender The spleen was palpated two fingers breadth below the *left* costal margin In addition a mass was palpable in the left upper quadrant which appeared to be independent of the spleen The Wassermann was 4 plus

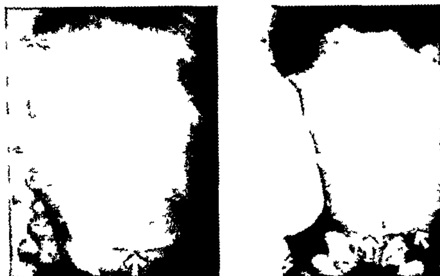


FIG 623 (A *Left*) Cyst of the spleen with calcification of the wall (B *Right*) Same patient as is shown in (A) Note the displacement of the stomach to the right by the cyst

At operation there were multiple small cysts throughout the liver Those on the surface were seen to contain clear fluid The kidneys were enlarged and polycystic There was a tremendous cyst of the spleen at least 8 inches in diameter adherent to the anterior abdominal wall the lateral wall the diaphragm above and the underlying peritoneum The cyst contained thick mucinous grayish white odorless material The spleen and the attached cyst were removed

The report of the macroscopic examination was *Specimen is a spleen containing a large cyst measuring 16 cm across The interior is shaggy and filled with dirty mucoid material The wall of the cyst is 3 by 6 mm and contains particles of calcium*

The report of the microscopic examination was Sections of the wall of the cyst show a thick, hyalinized connective tissue in which are collections of round cells and polymorphonuclear leucocytes The few blood vessels have thick walls Some of the cell collections may represent the remnants of compressed splenic pulp The cyst contents contain large quantities of cholesterol crystals stain with eosin and are acellular and amorphous No daughter cysts or *Taenia ecchinococcus* are seen The spleen with attached cyst wall

The diagnosis of cyst of the spleen in this case *depended on the following features*

1 The location of a mass in the left upper quadrant in the anatomical position of the spleen

2 The upward displacement of *the left* diaphragm

3 The displacement of the stomach to the right

4 Calcification of the wall of the mass

Areas of calcification may be present

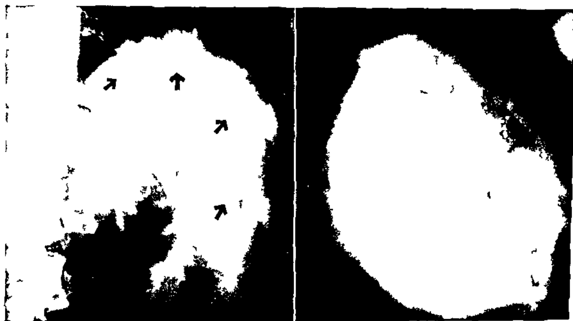


Fig 624 (A *Left*) Tubercles of the spleen (B *Right*) Same patient as is shown in (A) Roentgen appearance of the spleen removed at autopsy Note the small discrete areas of calcification

shows well defined malpighian bodies The trabeculations appear increased The capsule contains homosiderin and calcium

The diagnosis was cyst of the spleen

Roentgen examination (Fig 623A) revealed a large globular mass occupying the left upper quadrant and producing elevation of the left diaphragm The lower portion of this mass showed the presence of calcific plaques There was also considerable displacement of the stomach to the right by this mass (Fig 623B) It was believed that the mass was of splenic origin

As noted in the pathologic examination of the resected cyst the wall contained particles of calcium There was no evidence indicating that the cyst was of ecchinococcus origin

throughout the spleen as illustrated by the following well controlled case

J B male aged 37 The patient gave a 1 year history of weakness followed 4 months later by intermittent lower abdominal pain not related to food intake and occasional incontinence of feces and urine He lost 45 pounds Five months before his admission to the hospital *he developed a slight nonproductive* cough which persisted from then on

Physical examination revealed a large nontender mass above the left clavicle as well as numerous small lymph nodes above the mass and on the right side of the neck There was roentgen evidence of pulmonary tuberculosis Abdominal examination was essentially nega

tive A left supraclavicular node was removed

Pathologic diagnosis was tuberculosis of the lymph node

Autopsy revealed tuberculosis of the hilar and tracheobronchial lymph nodes tuberculosis of the trachea and bronchi tuberculous mediastinitis with ulceration into the esophagus fibrocaseous tuberculosis of the lungs and tuberculous pericarditis tuberculous ulcers of the ileum of the mesenteric nodes liver kidneys and spleen

The spleen weighed 380 Gm The capsule was studded with large soft white projections

The report stated that on section these areas appeared to be markedly caseous There was a small infarct of the spleen in the lower pole The splenic pulp appeared normal otherwise and many spicules of calcium could be palpated in the spleen

The pathologic diagnosis was tuberculosis of the spleen with calcification in the tuberculous foci

Roentgen examination (Fig 624A) showed numerous areas of calcification throughout the spleen varying in size shape and degree of density In view of the pulmonary tuberculosis and the tuberculosis of the supraclavicular node it was believed that these areas of calcification in the spleen were also tuberculous in nature This was confirmed at autopsy Figure 624B shows the appearance of the spleen on roentgen examination after removal at autopsy Note the disseminated areas of calcification

THE LIVER

The liver may be roentgenologically demonstrable even under ordinary conditions during routine examination of the abdomen



FIG 625 Cyst of the liver producing downward displacement of the transverse colon

There are a number of factors however which aid in demonstrating the liver more clearly (1) filling the colon with air may bring the liver shadow into clearer relief (2) following the administration of the gall bladder dye there may be an intensification of the liver shadow (3) thorium dioxide administered intravenously increases the radiopacity of the liver (4) pneumoperitoneum with films taken in different positions particularly erect and lateral will

show the liver contour with remarkable detail against the background of the air which envelops it In this manner the size of the liver and irregularities produced by cysts or tumors can be shown Thorium dioxide administration and pneumoperitoneum will be discussed more fully in connection with visualization of both liver and spleen

Cysts may also be demonstrable through

The diagnosis of cirrhosis of the liver may be substantiated by roentgen evidence of esophageal varices Similarly, if a primary malignant tumor of the liver is suspected, the demonstration of esophageal varices will be a material corroborative aid, because of the underlying cirrhosis so frequently present Thus, in an analysis of 24,400 autopsies at Bellevue Hospital from 1906 to 1936, Gustafson [9] found 62 primary



FIG 626 Echinococcus cyst of the liver (upper arrow) Note the dye filled gallbladder overlapping the spine (lower arrow)

calcification of the walls Abscesses and tumors of the liver may produce upward displacement of the right diaphragm with fixation of the diaphragm If the abscess contains gas, then a fluid level may be noted in the erect position and also with the patient placed laterally on his left side At times there may be associated pleural effusion just above the diaphragm Tumors of the liver may also be demonstrable by displacement of neighboring viscera such as the stomach and duodenum and by displacement of the right half of the colon as well

carcinomas of the liver 32 or 51 6 per cent, were associated with cirrhosis

In a later report of Peller,[30] based on 6 596 autopsies at Bellevue between 1934 and 1941, of 5 988 individuals without cirrhosis, there were 22 malignant tumors of the liver In 608 individuals with cirrhosis there were 20 carcinomas obvious evidence of the susceptibility of the cirrhotic to the development of primary intrahepatic malignancy

The demonstration of esophageal varices therefore offers support for the diagnosis of cirrhosis of the liver and is only indi



Fig 627 Echinococcus cysts of the liver

rectly an indication of the pathologic nature of the engrafted tumor

Illustrative Cases A tumor of the liver may cause downward displacement of the transverse colon

H M female aged 64 During the preceding year and 4 months the patient noticed a progressive painless enlargement of the abdomen Physical examination revealed a cystlike mass occupying almost the entire upper abdomen

Operation revealed a huge cyst involving the liver On puncturing the cyst 7 800 cc of a thin chocolate colored fluid was evacuated There was no evidence of infection or calcification The cyst wall on the inside was perfectly smooth A portion of the cyst wall was removed for pathologic examination

The diagnosis was bile duct cyst

Roentgen examination (Fig 625) revealed evidence of a huge mass involving almost the entire upper abdomen and causing downward displacement of the transverse colon As determined on pathologic examination the tumor was a huge bile duct cyst of the liver

The roentgen appearance of echinococcus cyst of the liver is illustrated by the next case

C D male aged 49 About 2½ years before his admission to the hospital this patient was seized with an attack of severe right upper quadrant pain radiating along the lower right costal margin After about 4 days of intermittent pains he became jaundiced and had clay colored stools The jaundice lasted for approximately 2 months From that time on the patient had attacks of pain in the right upper quadrant associated with jaundice There were no vomiting chills or fever

Physical examination of the abdomen revealed the liver palpable three fingers breadth below the costal margin There was a tender mass in the right upper quadrant and a similar mass in the left upper quadrant

At operation two cysts of the liver were found One was high in the dome of the right lobe with a smooth hard capsule about 1½ inches in diameter The second cyst was in the right lobe laterally, below the costal margin it was about 2 inches in diameter slightly umbilicated and with a dense hard capsule The gallbladder was normal The common duct was dilated to about the diameter of the fifth finger No stones or daughter cysts were found in the common duct The lower cyst of the liver was opened The cavity was thoroughly swabbed with 25 per cent formalin and its contents were evacuated The cyst was then packed

Microscopic examination of sections through the cyst wall revealed a homogeneously stained tissue lined by an irregular, amorphous material having a granular appearance Sev



Fig 628 Calcified area in the liver

eral isolated polymorphonuclear leukocytes were seen No hooklets were noted

The diagnosis was "Although no hooklets are seen the lining of the cyst wall is compatible with an echinococcus cyst"

Roentgen examination (Fig 626) revealed an irregularly outlined mass with a calcific rim at the lateral border of the liver at the lower costal margin In addition, a similar lesion not shown in this roentgenogram, was found high up under the dome of the liver The gallbladder was also visualized and showed no evidence of organic disease as corroborated at operation

mass in the right upper quadrant moving with respiration

The autopsy report was 'Far advanced pulmonary tuberculosis On opening the abdomen, the omentum is adherent to a hard irregular mass approximately 6 cm in diameter which lies in the lower margin of the right lobe of the liver The region normally occupied by the gallbladder contains a large calcified mass which when opened is found to contain bile and to communicate directly through a fistulous opening into the first portion of the duodenum On the posterior surface of the right lobe of the liver is a hard



Fig 629 Abscess of the liver with upward displacement of the diaphragm

The final diagnosis was echinococcus cyst of the liver with calcific rim

Multiple areas of calcification of the liver, the result of echinococcus cysts may be noted in the following case

M C male aged 49 Seven months before his admission to the hospital the patient complained of weakness and easy fatigue and a nonproductive cough night sweats and a loss of 20 pounds in weight He had known for many years that there was a lump in his upper abdomen

Physical examination revealed an advanced pulmonary tuberculosis In addition there was a large firm irregularly outlined nontender

white mass measuring approximately 2½ cm in diameter which when opened is found to have a calcified wall approximately 4 mm thick and to contain laminated irregular masses of gelatinous gray and yellow material Occupying most of the left lobe of the liver is another similar but larger calcified mass measuring approximately 6 cm in diameter and having similar contents

The final diagnosis was echinococcus cysts of the gallbladder and liver

Roentgen examination (Fig 627) revealed a mass with a calcified rim in the region of the right lobe of the liver near its lower border In addition there were some other irregularly outlined calcific structures within this region High up in the hepatic area was

a small rounded area with a calcific rim There was another ovoid structure about the size of a walnut in the region of the left lobe of the liver, partially calcified particularly along the rim

vomiting The pain ultimately became more severe and constant with radiation to the back

Two and one half months before his admission to the hospital, he vomited coffee



FIG 629A Hepatic abscess Note (1) marked enlargement of the liver shadow (2) marked upward dislocation of the right diaphragm (3) irregularly outlined translucent areas within the confines of the hepatic shadow Note also the calcified splenic artery in the left upper quadrant of the abdomen

The findings were those of calcified echinococcus cysts of the liver

A calcified area in the liver of undetermined origin is described in the next case

M di G male, aged 55 During the preceding 1½ years the patient had had recurring attacks of abdominal pain about 1 to 1½ hours after meals at times relieved by

ground material and had tarry stools While on the ward the patient had a sudden sharp pain just above the umbilicus which spread all over the abdomen

Physical examination of the abdomen revealed boardlike rigidity and an absence of liver dullness

Operation revealed a perforated gastric ulcer A large quantity of gray, dirty fluid,

including food particles was noted The perforation was about 1¼ by ½ cm and was present on the anterior surface of the lesser curvature of the pars cardia

Prior to the operative intervention, roentgen *examination of the gastro intestinal tract* had been made and, incidentally, an irregularly rounded calcific shadow was found high up under the right diaphragm, apparently in the liver substance (Fig 628)

At autopsy, in addition to the evidence of a perforated gastric ulcer and peritonitis, an examination was made of the liver and in the dome on the right side there is encountered a calcified round nodule measuring approximately 2 cm in diameter The remainder of the liver shows no evidence of gross pathology '

The exact nature of this calcific area was not determined

An abscess of the liver may cause upward displacement of the diaphragm

M C, female aged 28 The patient had been entirely well until 2 weeks before her admission to the hospital when she first noted a pain in her right chest which gradually increased in severity She had fever vomiting and exaggeration of the pain on breathing On examination of the right chest an area of flatness to percussion was found over the fifth, sixth, and seventh interspaces between the anterior axillary line and the sternal line There was considerable tenderness on palpa*tion under the right rib but no spasm While* under observation she developed pain in the epigastrium and right upper quadrant and in the region of the right shoulder A prominence of the right lower anterior ribs developed Examination of the stool showed no evidence of parasites A blood specimen was tested for echinococcus antibody by the complement fixation test and was found to be definitely negative



FIG 630 Cirrhosis of the liver Visualization of the liver and the spleen after administration of thorotrast

At operation there was a large mass in the dome of the right lobe of the liver with firm adhesions between the liver and the diaphragm at this site The mass on palpation was approximately the size of a small orange *A needle was introduced into the mass and 15 to 20 cc of very thick, yellow pus was* withdrawn The abscess was opened and drained

Roentgen examination (Fig 629) showed the localized elevation of the right diaphragm produced by the abscess

An abscess of the liver may be associated with irregularly outlined translucent areas

which are apparently due to associated gas formation

G F, female aged 76 The patient entered the hospital as a case of unexplained fever varying between 102 and 105, with failure of response to massive doses of penicillin Examination of the abdomen was essentially negative During her stay at the hospital she developed evidence of pneumonia involving the right upper lobe Subsequent blood cultures were positive for the Friedlander bacillus

At autopsy, examination of the liver revealed it to be adherent to the anterior chest wall and right dome of the diaphragm The liver weighed 2 300 Gm The right lobe felt as if it had a large fluctuant mass Section revealed a large abscess 8 by 10 by 15 cm occupying most of the lobe and filled with thick green purulent material lined by a capsule of fibrous tissue from 1 to 2 mm thick

In addition a lobar pneumonia was present involving the right lung with a right pleural effusion and empyema The culture showed that the pulmonary lesion and the abscess of the liver were due to the Friedlander bacillus In addition to other pathologic lesions of a more minor character it was noted that the splenic artery showed marked calcification throughout its entire length

Roentgen examination (Fig 629A) of the abdominal cavity made with the portable bedside unit revealed the following

1 Marked enlargement of the liver shadow

2 Marked upward dislocation of the right diaphragm

3 Irregularly outlined translucent areas within the confines of the liver shadow

Examination in the erect position could not be made

Based on these findings a diagnosis of a gas containing abscess of the liver was made In addition a calcified vessel may be noted to the left of the spine in the direction of the spleen This had the characteristics of a calcified splenic artery as confirmed at autopsy



FIG 631 Visualization of the liver after thorotrast Subacute hepatitis The patient died of carcinoma of the pancreas

THOROTRAST IN THE VISUALIZATION OF LIVER AND SPLEEN

Owing to the tendency of thorotrast to be deposited in the reticulo endothelial cells the spleen and liver may be visualized following the intravenous injection of this substance The use of thorotrast for such

purposes was introduced by Oka[31] and Radt[32]

Thorium dioxide is administered in the form of a colloidal solution The amount usually administered is 75 cc divided into three doses of 25 cc each and administered intravenously on three successive days Roentgen exposures are made the following day

The procedure has been applied to the localization of tumors suspected of being in the liver and spleen, to the diagnosis of atrophic and hypertrophic cirrhosis of the liver, to abscess cysts and amyloidosis of the liver In atrophic cirrhosis the liver may be small and diffusely mottled in hypertrophic cirrhosis the liver is enlarged and usually casts a homogeneous shadow of diminished density In the case of tumors corresponding to the regions of involvement, which are ordinarily devoid of reticulo endothelial cells and are therefore free of thorium, there are rounded more or less translucent areas Its application to the demonstration of metastatic invasion of the liver is probably the most valuable contribution that this procedure has to make

Because of the extreme importance of the reticulo endothelial cells considerable caution should be employed in the use of this technic for the purpose of the roentgen visualization of the liver and spleen Thorotrast imbedded in these cells may interfere with their function Moreover, the substance is not eliminated even over a period of years In spite of the apparent possibilities of danger which might result from the presence of a radioactive substance in the liver and spleen over a long period of time, however, there are a number of essentially favorable reports on its use[33-36]



Fig 632 Visualization of the spleen after pneumoperitoneum

Serious objection to the use of thorotrast has been made by other workers in this field Shih and Jung[37] injected thorotrast intravenously into rabbits employing 0 5 cc per Kg on 2 consecutive days Four rabbits were given larger doses One of the rabbits, a pregnant female which had received the smallest dose aborted after the first injec

tion In others even with the small dose there was evidence of reduction of blood platelets and petechial hemorrhages in the lungs and kidneys The conclusion of the investigation was that thorium given intravenously tends to produce acute purpura hemorrhagica

In one of the cases reported by Stewart Einhorn and Illick[38] in which the liver had been made more opaque by the injection

thereby affect the body s immunity mechanism In addition, they observed damage to the liver and spleen Because of the radioactive nature of the substance, cumulative radioactive effects may develop, possibly of a dangerous nature

The Council on Pharmacy and Chemistry of the American Medical Association summarized its findings in an investigation of thorotrast as follows



Fig 633 (A *Left*) Air under both diaphragms after artificial pneumoperitoneum (B *Right*) Same patient as is shown in (A) Enlarged liver shown after pneumoperitoneum

of thorotrast the spleen was removed at autopsy Placed on a photographic plate for a day the spleen contained enough thorium to produce an image although in control studies the normal spleen failed to do so In four cases injected with thorotrast that came to autopsy there was an acute splenitis

Orr Popoff Rosedale and Stephenson[39] from their experiments with thorotrast concluded that the substance should not be injected into human beings because of failure of elimination from the body it may block the reticulo-endothelial system and

In view therefore of the very imperfect elimination of thorium dioxide its fairly high alpha ray activity the possibility of further increase in radioactivity by partial conversion to mesothorium and radiothorium and the possibility of sensitization of tissues to roentgen rays considering the short period during which patients have been kept under observation the Council voted that thorotrast be not accepted for intravenous administration

In view of the apparent latent dangers inherent in this procedure evidence of which might conceivably be obscured for a number of years the method of investiga

tion of the liver and spleen through the intravenous administration of thorotrast is *therefore not recommended* and at the present time this method is used at Bellevue Hospital only under the most exceptional circumstances

An example of the effectiveness of thorotrast in the visualization of the liver and spleen is shown by the case of G R, who died with evidence of cirrhosis of the liver *Figure 630 shows the liver as a dense* shadow sharply defined The enormously enlarged spleen is also beautifully shown in *the roentgenogram*

Figure 631 shows a small homogeneously dense liver with sharply defined contour after the injection of thorotrast in an individual who died of a carcinoma of the pancreas At autopsy the liver was found to be smaller than normal Microscopic examination showed a subacute hepatitis No metastases were present in the liver For some unexplained reason the spleen shows only a very moderate increase in density The roentgen findings produced by the carcinoma of the pancreas are described under the subject of pancreatic tumors

PNEUMOPERITONEUM IN THE VISUALIZATION OF THE LIVER AND SPLEEN

At first, air was injected into the peritoneal cavity *so as to facilitate the* introduction of instruments for direct visualization of intra abdominal viscera [40-42] *Following these attempts at direct inspec*tion of the viscera within the abdomen roentgen examination was carried out on *the abdomen after it had been previously* distended with air [43-47] The procedure of pneumoperitoneum undoubtedly produces beautiful roentgenograms *for the visualiza*tion of the viscera and of abnormalities within the abdomen, such as tumors of various sorts either retroperitoneal or originating from any of the abdominal viscera Enlarged lymph nodes have been described and adhesions particularly to the abdominal wall have been clearly demonstrated It also may be used as an aid



Fig 634 A Adhesions of the liver to the diaphragm shown by pneumoperitoneum

in the differential diagnosis between herniation of the stomach and eventration, as previously described

Technic

The following technic is employed in pneumoperitoneum The patient is given a cleansing enema and the urinary bladder is emptied A hypodermic of morphine may be administered shortly before puncture The point for puncture is about 1½ inches to the left of the midline and below the level of the umbilicus A lumbar puncture needle with its stylet is introduced slowly until one feels that a cavity has been entered At the time of perforating the peritoneum the patient will usually complain of severe pain Atmospheric air is then introduced through the needle by means of a Potaine aspirator A gurgling sound will be heard on auscultation as the air enters the abdominal cavity About 1 000 cc of air are usually introduced following which roentgenologic exposures are made in various positions which include antero-posterior and postero-anterior views the right and left lateral decubitus and right and left lateral prone positions and the sitting position In the event of considerable discomfort the needle may then be reintroduced and some of the air permitted to escape

The dangers are on the whole negligible puncture of the intestine infection gas embolism and the formation of adhesions being of very infrequent occurrence However when the usual diagnostic procedures have failed and a surgical condition is suspected an exploratory laparotomy with a larger abdominal opening under the most aseptic technic may in many cases be preferable to the use of artificial pneumoperitoneum

Illustrative Cases An example of clear visualization of the spleen after pneumoperitoneum is to be seen in figure 632 Note the presence of the air outlining the left diaphragm

Figure 633A shows the subdiaphragmatic collection of air in a case of artificial pneumoperitoneum Figure 633B in the same case shows an enlarged but smoothly outlined liver

Pneumoperitoneum may demonstrate adhesions of the liver to the undersurface of the diaphragm as illustrated by the following case

W T male aged 62 This patient entered the hospital complaining of an abscess over



Fig 634 B This is the same patient as is shown in Figure 634 A Note the lipiodol injected into the sinus

the right thorax of 5 months duration On the morning of admission he first noticed that the abscess was discharging from a small sinus

Physical examination revealed a mass about 6 inches in diameter over the lateral aspect of the right thorax A sinus was present from which a thin yellowish pus exuded In the axillary region on the right side there was another mass irregular firm and slightly tender

The report of operation was as follows The abscess was incised and the cavity was packed The sinus continued to discharge There was no pain Reoperation revealed an encapsulated abscess involving the supra

phrenic region on the right side with a spontaneous fistula pointing in the midline

Roentgen examination after pneumoperitoneum (Fig 634A) showed a dense, oblique shadow, extending from the middle of the dome of the diaphragm outward and upward to the axilla The outer half of the dome of the diaphragm and of the costophrenic sinus *were completely obliterated* Pneumoperitoneum distinctly showed the inner half of the dome of the diaphragm Lipiodol injection showed a sinus extending to the region of the dome of the diaphragm (Fig 634B) The spleen was well outlined and normal in appearance

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65

Roentgen Diagnosis of Diseases of the Pancreas

PANCREATIC TUMORS
PANCREATIC FIBROSIS
PANCREATIC CALCULI
PANCREATIC ABSCESS

When the pancreas is enlarged by cysts or tumors, it may often be recognized by secondary manifestations such as displacement or compression of neighboring viscera. A tumor of the head of the pancreas may cause enlargement of the duodenal curve, with displacement to the right of the second portion and downward displacement of the transverse portion of the duodenum. In addition there may be an upward displacement of the distal portion of the stomach.

A tumor of the head of the pancreas may so compress the first portion of the duodenum as to lead to marked gastric enlargement and abnormal retention, roentgenologically simulating an obstructive lesion of the pyloroduodenal region of intrinsic origin. It may also invade the lumen of the duodenum. A tumor, however, may be present without roentgen evidence of its presence under three conditions. (1) The tumor may be too small to cause recognizable evidence of displacement of neighboring viscera, although capable of causing serious clinical derangements. (2) The tumor may grow primarily in a posteroanterior direction so that there is no mechanical pressure exerted upon the duodenal curve. (3) The duodenal curve may show various types of congenital anomaly in its course, so that the head of the pancreas does not sit in the lap of the duodenal curve. The pancreatic growth will therefore be unable to expand the curve of the duodenum.

In addition to displacement, the irregular growth of the tumor may cause compression of various portions of the duodenum so that it fills in an irregular manner. Gutmann and Jahiel[1] described two concave depressions of the inner border of the second portion of the duodenum, one above and one below its midportion, as evidence of carcinoma of the head of the pancreas. This finding was more fully elaborated by Frostberg, who described the deformity as resembling a reverse 3. The growth may cause downward displacement of jejunum and transverse colon.

By adhering to the posterior wall of the stomach, the tumor may cause a diminution of gastric mobility. Finally, it may actually ulcerate through into the lumen of the stomach or duodenum. The roentgen picture of the stomach may then be indistinguishable from that of an intrinsic gastric lesion.

As with retrogastric tumors generally, a mass of pancreatic origin, particularly of the body or tail, may produce forward displacement of the stomach as well as a pressure defect on the posterior wall. As previously described in connection with the subject of extragastric tumors (see Chap. 17), the examination is preferably conducted with the patient in the erect left lateral position before and after the ingestion of barium. The roentgenogram prior to the administration of barium may show the circumscribed mass. With the stomach filled with barium, one may note unusual forward displacement as well as a pressure defect on the posterior wall.

A tumor of the body of the pancreas may al o displace the stomach either upward or downward, depending on its relative anatomical location When present in the tail it may displace the stomach to the right A calcified echinococcus cyst of the tail of the pancreas was described by Valenti[3]

Because of the pressure upon the common duct by a pancreatic tumor there may be marked enlargement of the gallbladder demonstrable in the scout film or after the administration of the gallbladder dye When filled with the dye the gallbladder may then show abnormal delay in emptying

Illustrative Cases Enlargement of the duodenal curve as an evidence of tumor of the head of the pancreas is illustrated by the following case

J S aged 45 Four weeks before admission to the hospital this patient developed pain in the left upper quadrant it was continuous and burning and without radiation or relation to meals There was no vomiting jaundice or clay colored or tarry stools

The report of the physical examination was Two inches above the umbilicus is a freely movable mass the size of a fist tender to palpation The surface is irregular The liver is palpable 9 cm from the costal margin in the parasternal line and is firm and smooth

Operation revealed a carcinoma of the head of the pancreas

Roentgen examination (Fig 635) showed considerable enlargement of the duodenal curve with a very irregular distribution of the barium through it The appearance was that of a tumor of the head of the pancreas causing enlargement of the duodenal curve The irregular distribution of the barium through the duodenal curve was apparently due to the irregular nature of the growth



FIG 635 Tumor of the head of the pancreas showing *enlargement of the duodenal curve*

A tumor of the head of the pancreas may produce a reverse 3 deformity of the inner border of the second portion of the duodenum

I F male aged 64 During the preceding 6 months the patient had complained of intermittent pain in the right upper quadrant not related to food intake There was occasional vomiting

On physical examination there was definite icterus No definite mass was palpable on abdominal examination Liver chemistry studies pointed to an obstructive jaundice

65

Roentgen Diagnosis of Diseases of the Pancreas

PANCREATIC TUMORS
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In addition to displacement, the irregular growth of the tumor may cause compression of various portions of the duodenum, so that it fills in an irregular manner Gutmann and Jahiel[1] described two concave depressions of the inner border of the second portion of the duodenum one above and one below its midportion, as evidence of carcinoma of the head of the pancreas This finding was more fully elaborated by Frostberg[2] who described the deformity as resembling a reverse 3 The growth may cause downward displacement of jejunum and transverse colon

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Although the diagnosis of carcinoma of the head of the pancreas was not established by pathologic examination a definite tumor was present in the head of the pancreas and the clinical probability was very strong that this tumor represented a carcinoma

Roentgen examination (Fig 637) showed a concavity above and below the midportion of the inner border of the second part of the duodenum This had the appearance of an inverted 3 sometimes associated with the presence of a tumor of the head of the pancreas The duodenal bulb showed a deformity due to the old ulcer which had been found 5 years before

That part of the duodenum between the upper and the lower concavities probably represents the papilla of Vater which may exhibit greater resistance to the pressure of a mass in the head of the pancreas than the duodenum proximal and distal to it This may explain the peculiar nature of the deformity produced by the pancreatic tumor In this sense the pressure deformity of the duodenum may be somewhat analogous to that produced by the erosion of a vertebra by an abdominal aneurysm which leaves intact the more resistant intravertebral disk

A tumor of the head of the pancreas may not only produce pressure deformities at the inner border of the duodenum but may actually invade its lumen

B A male aged 43 This patient had been in good health until a year before his admission to the hospital when he began to have right upper quadrant discomfort and to show gradual loss of weight These symptoms became progressively worse Fatty foods increased the pain

Operation (elsewhere) revealed a large firm diffuse tumor of the head of the pancreas involving also the body There were two umbilicated metastatic nodules visible on the surface of the liver



Fig 638 Carcinoma of the head of the pancreas invading the second portion of the duodenum

About 2 months later the patient applied for admission to Bellevue Hospital At this time a hard mass was palpable in the right upper quadrant The mass was exquisitely tender to palpation The liver edge was palpable two fingers breadth below the costal margin While at the hospital he had tarry stools on a number of occasions No further operative interference was attempted on this patient

Roentgen examination (Fig 638) revealed irregularly outlined translucent areas within the second portion of the duodenum There was destruction of the normal mucosal markings in this region This area appeared moderately dilated The appearance was that of a



Fig 636 Carcinoma of the head of the pancreas Note the reverse 3 at the inner border of the second portion of the duodenum

At operation, the gallbladder was markedly distended but emptied on pressure The common duct was dilated Nodes were palpable in the duodenohepatic ligament and gastrohepatic ligament The head of the pancreas was enlarged and was found to be hard and nodular There were several hard umbilicated nodules in the liver which were definitely malignant

The diagnosis was carcinoma of the head of the pancreas with metastases to the surrounding nodes

A cholecystgastrostomy was done

A biopsy of the liver was taken and a lymph node removed

The pathologic diagnosis was adenocarcinoma in lymph node and liver

Roentgen examination (Fig 636) showed two concave defects at the inner border of the second portion of the duodenum giving the appearance of a reverse 3 The mucosal structure in the distal part of the second portion of the duodenum was abnormal in appearance There was also evidence of pressure on the pars pylorus The reverse 3 appearance the abnormal mucosal pattern of the duodenum and the pressure defect of the pars pylorus supported the diagnosis of a tumor in the region of the head of the pancreas probably invading the duodenum As noted at operation, the patient had a malignant tumor of the head of the pancreas The lumen of the duodenum had not been examined

The significance of the reverse 3 appearance of the second portion of the duodenum is further illustrated by the next case

N D, male aged 66 The patient had had a gastroenterostomy for duodenal ulcer with marked obstruction 5 years before Four months prior to his admission to the hospital he developed dull pain in the upper abdomen and back which occured in the midafternoon lasting for about ½ hour and relieved by food The pain gradually became worse the urine dark the stools clay colored and he sought admission to the hospital He had lost 45 pounds in 3 months

Physical examination revealed deep jaundice The liver was palpated two fingers breadth below the costal margin and a mass was palpable below the liver edge Diabetes was present After controlling the diabetes a laparotomy was done

Operation disclosed a lemon sized firm mass in the head of the pancreas A cholecystojejunostomy and jejunojenostomy was done in preparation for a second stage removal of the tumor This second operation however was not done because the patient s general condition although he was relieved of jaundice precluded any further surgery



Fig 637 Carcinoma of the head of the pancreas Note the reverse 3 at the inner border of the second portion of the duodenum

which became progressively deeper There was no blood in the vomitus or stool The main features on physical examination were the deep jaundice and a large hard mass in the right upper quadrant

Operation revealed a globular stony hard mass occupying the entire head and body of the pancreas 4 inches in diameter protruding forward so that the stomach draped itself over this mass The gallbladder was tensely distended 5 inches long and 3 inches in diameter There was a nodule in the left lobe of the liver no other obvious metastases A cholecystgastrostomy was done

Roentgen examination (Fig 640) revealed a crescentic pressure defect of the greater curvature of the pars pylorus and the duodenal bulb This was interpreted as having been produced by a tumor of the pancreas



Fig 641 Carcinoma of the body and the tail of the pancreas producing irregular narrowing of the prepyloric portion of the stomach

The deformity of the stomach produced by the pressure of a carcinoma of the pancreas may in rare instances be indistinguishable from a primary gastric lesion

F P male aged 81 The patient gave a 2 month history of weakness epigastric pain and a weight loss of 35 pounds On physical examination the liver was felt two fingers breadth below the right costal border apparently continuous with a large hard rough sharpedged mass projecting downward from the epigastrium to below the umbilicus No jaundice was present There were many subcutaneous nodules varying in size from that of a pea to that of a hazel nut firm and non tender A subcutaneous nodule was removed from the left axilla The pathologic diagnosis was metastatic adenocarcinoma primary site unknown

At autopsy there was an adenocarcinoma of the body and tail of the pancreas with extension to the head and cystic degeneration of the body There was involvement of the para aortic and mesenteric nodes liver right lung the first left rib right kidney peritoneum subcutaneous tissues and both adrenals The entire peritoneum was studded with small white nodules 1 500 cc of clear amber fluid was present in the abdominal cavity The head of the pancreas was of usual consistency Three cm to the left of the duodenum the pancreas was occupied by a large cavity extending up to compress the pre



Fig 639 Tumor of the pancreas, *showing the upward dislocation of the* stomach

tumor within the second portion of the duodenum

As determined at operation, this tumor had evidently infiltrated the duodenum from the pancreas The tarry stools observed in this case also indicate the involvement of the duodenum

Upward dislocation of the stomach by a tumor of the pancreas is well shown by the next case

J R male, aged 59 Four weeks before his admission to the hospital this patient first complained of pain in the abdomen *which occurred 1 hour after eating his supper* The pain continued all night From then on the pain persisted with varying severity It was cramplike and was exaggerated by the ingestion of food He had lost 25 pounds in the preceding year There was no jaundice tarry stool vomiting or disturbance of in testinal function

Operation revealed a carcinoma of the pancreas The pancreas was enlarged and nodular throughout There was massive nodular involvement of the liver The gall bladder was dilated Nodes at the hilum about *the common duct* over the pancreas and in the gastrocolic omentum were enlarged and hard Free straw colored fluid was present in the peritoneal cavity *The stomach and duo* denum were negative

Roentgen examination (Fig 639) revealed no intrinsic organic lesion of the stomach or duodenum The pylorus and duodenum were directed downward in almost perpendicular fashion The duodenal curve was enlarged *These findings* were explained on the basis of a tumor in the region of the pancreas causing upward displacement of the midportion of the stomach with secondary displacement down ward of the pyloroduodenal region and en *largement of the duodenal curve As noted* above, the presence of a carcinoma of the head of the pancreas was confirmed at opera tion

A carcinoma of the pancreas may pro duce a marked pressure defect on the greater curvature of the pylorus and the duodenal bulb

A C male, aged 53 The patient gave a 5 week history of frequent bowel movements (every 15 to 20 minutes) The stools were *white in color and hard One week later he* complained of localized pain in the right upper quadrant He vomited on a few occa sions and lost 30 pounds Shortly before his admission to the hospital he noticed jaundice



Fig 640 Carcinoma of the head of the pancreas Note the pressure defect on the greater curvature border of the pars pylorica and the duodenal bulb

pyloric region of the stomach It contained turbid brown yellow fluid The tail was stony hard with complete absence of lobular structure The stomach and duodenum were normal Microscopic examination showed carcinoma throughout the pancreas

Roentgen examination (Fig 641) showed narrowing and irregularity of the prepyloric portion of the stomach on the basis of which a diagnosis of carcinoma of the pylorus was made As shown by autopsy study the deformity was the result of compression by the pancreas

A tumor of the body of the pancreas may produce a pressure defect on the lesser curvature of the stomach

C D, male aged 71 During the preceding 9 months the patient complained of epigastric pain not related to meals and occasional nausea and vomiting The pain occasionally radiated to the lumbar spine He had never been jaundiced nor were the stools ever clay colored He lost 30 pounds in 5 months Physical examination revealed a tender nodular epigastric mass

The report at autopsy was There was a carcinoma of the body of the pancreas with secondary carcinoma in the periportal and retroperitoneal lymph nodes liver gall bladder left adrenal and peritoneum The esophageal and gastric mucosa were normal The serosa of the cecum and that of the appendix was sprinkled with small pearl like tumor tissue implants

Roentgen examination (Fig 642) showed a smooth pressure defect on the lesser curvature of the stomach pars cardia and media evidently produced by the palpable tumor mass As noted at autopsy the tumor was a carcinoma of the body of the pancreas

A carcinoma of the pancreas may fix the stomach posteriorly Also it may displace the loops of small intestine downward Both these features are illustrated by the next case

J F aged 64 For 3 months this patient complained of distress in the epigastrium occurring after eating The pain was worse at night It was relieved by aspirin but not by soda Forced vomiting produced relief

Physical examination revealed tenderness in the upper abdomen particularly in the epigastrium and right upper quadrant On deep palpation an elongated mass about the size of the little finger was felt through the abdominal wall in the right upper quadrant There was no evidence of jaundice

At operation there was a large mass of what appeared to be carcinoma in the great omentum The gallbladder was enormously distended There was a large retroperitoneal hard tumor which extended along the superior



Fig 644 Carcinoma of the tail of the pancreas invading the lumen of the pars cardiaca of the stomach

border of the pancreas and was adherent to the posterior wall of the stomach near the lesser curvature

Roentgen examination (Fig 643) revealed the following The stomach appeared fixed high in the abdomen There was no irregularity of contour A smooth crescentic pressure defect was noted displacing the coils of jejunum downward



Fig 642 Carcinoma of the body of the pancreas producing pressure on the lesser curvature of the pars cardia and pars media of the stomach



Fig 643 Downward displacement of the loops of the small intestine by a tumor of the pancreas



FIG 646 C Same patient as is shown in Figure 646 A and B Examination in the left lateral supine position with the stomach containing barium Again note the anterior displacement of the stomach and the pressure defect on the posterior wall

veloped pain in the abdomen it was intermittent and cramplike The stools were occasionally black There was jaundice He vomited on several occasions the vomitus contained no blood but was bile stained He lost 30 pounds in weight

Physical examination of the abdomen revealed no definite palpable masses Diffuse generalized tenderness was present

At operation there was a large mass occupying the posterior aspect of the cardiac area of the stomach with firm fixation posteriorly

The autopsy report was as follows The stomach duodenum liver pancreas omentum and the peritoneum covering the anterior abdominal wall were closely adherent to one another by thick fibrous adhesions They were separated from the anterior abdominal wall and removed in toto The stomach and duodenum were opened and found to be full of dark red partially clotted blood After removing this a fungating mass 7 cm in diameter was found perforating the posterior wall of the stomach it was brownish red and very friable

Microscopic examination of the pancreas shows that the normal architecture of the pancreas is completely destroyed There is



FIG 646 D Same patient as is shown in Figure 646 C Examination in the erect left lateral position Note the forward displacement of the stomach and the pressure defect on the posterior wall



FIG 645 Occlusion of the pyloroduodenal region by carcinoma of the pancreas



FIG 646 (A *Left*) Carcinoma of the tail of the pancreas Note the evidence of a soft tissue mass between the greater curvature of the stomach and the descending colon (B, *Right*) Same patient as is shown in (A) Left lateral supine position showing the tumor producing anterior displacement of stomach artificially distended with air

The conclusion was that the high fixation of the stomach and the smooth crescentic pressure defect of the coils of small intestine might be explained by the presence of a retroperitoneal tumor in the region of the pancreas fixing the stomach posteriorly and displacing the coils of small intestine downward This was verified surgically, as noted above

Occasionally a carcinoma of the pancreas may infiltrate and invade the lumen of the stomach, as illustrated by the following case

J C male aged 59 Five months before his admission to the hospital this patient de-

J K male aged 72 Two weeks before his admission to the hospital this patient noticed that his legs and abdomen had begun to swell He also complained of shortness of breath He had diarrhea during this period There was considerable loss of weight There was no pain at any time

Physical examination revealed the abdomen to be considerably distended with fluid The liver was palpable about two fingers breadth below the costal margin A moderate degree of jaundice was present Following paracentesis a mass was palpable about 3 inches to the right of the midline near the costal margin It was soft in consistency and about the size of an orange

Autopsy revealed a carcinoma involving the head of the pancreas about 5 cm in diameter which almost completely constricted the common duct The gallbladder was greatly distended The duodenum showed evidence of ulceration and carcinomatous infiltration secondary to the carcinoma of the pancreas

Roentgen examination (Fig 645) revealed the stomach to be of considerably increased size There was a total gastric residue at 6 hours after the ingestion of the barium The duodenum was not visualized at any time during the course of the examination The roentgen diagnosis was pyloric obstruction of organic origin As noted above the cause of the obstruction was the compression produced upon the duodenum by the malignant infiltration from the pancreas

A carcinoma of the pancreas may produce anterior displacement of the stomach similar to retrogastric masses generally The left lateral position is then of considerable value in localizing the position of the tumor This is illustrated by the following case



Fig 648 Downward displacement of the colon by a pancreatic cyst

J K male aged 56 The patient was well until 4 days prior to his admission to the hospital when he developed generalized abdominal pain constant from then on associated with vomiting and marked constipation There was no blood in the vomitus or stool There was a weight loss of 9 pounds

Physical examination revealed generalized abdominal tenderness most marked in the left upper quadrant where a mass was palpable

At operation there was a carcinoma of the midportion of the pancreas with metastases The retrogastric tumor was from 9 to 10 cm in diameter hard and fixed to the greater curvature of the stomach the gastrocolic omentum and the transverse colon

Roentgen examination (Fig 646A) showed

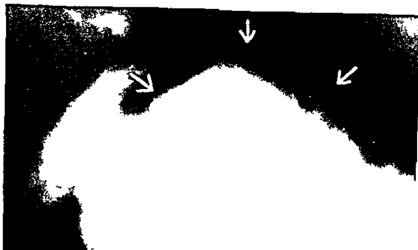


FIG 646 E Same patient as is shown in Figure 646 D Examination in the left lateral, supine position, without the introduction of air or barium into the stomach shows the soft tissue shadow produced by the tumor



FIG 647 Upward displacement of the stomach and enlargement of the duodenal curve by a retroperitoneal tumor

atrophy of the parenchymal cells and replacement with great quantities of hyalinized tissue which is infiltrated by small nests of anaplastic polyhedral cells"

The diagnosis was scirrhous carcinoma of the pancreas

The report of the microscopic examination of the stomach was 'Sections of the stomach in the region of the perforation described in the gross show destruction of the muscularis, submucosa and mucosa and infiltration of the adjacent areas by great quantities of connective tissue containing numerous nests of anaplastic polyhedral cells and polymorphonuclears

The diagnosis was 'extension of carcinoma of the pancreas into the stomach wall with perforation

Examination of the liver showed that it was smaller than normal, weighing 1,050 Gm No metastases were found (see Fig 631) Microscopic examination of the liver showed evidence of a subacute hepatitis

Roentgen examination (Fig 644) showed absence of the normal mucosal markings in the pars cardia and the pars media of the stomach and displacement of barium within this region On fluoroscopic examination when the patient drank the barium in addition to the deformity of the air bubble, there was a splitting of the stream of barium as it left the esophagus and entered the stomach as if encompassing a mass within it The diagnosis was of a new growth within the cardiac end of the stomach

As indicated by the autopsy findings this was due to an actual breaking through into the lumen of the stomach by a carcinoma originating in the tail of the pancreas

The next case is one of carcinoma of the *pancreas that produced almost complete* occlusion of the duodenum

the region of the head of the pancreas As determined at operation and biopsy examination the retroperitoneal mass was of lymphoid origin

A cyst of the pancreas may produce displacement of neighboring structures as is shown by the following example

F P female aged 25 This patient gave a history of 1 year s duration of a mass in the upper abdomen which had gradually increased in size Her only symptoms were a feeling of fullness in the abdomen nausea vomiting and increasing constipation

On physical examination there was a mass in the upper abdomen which appeared to be fixed and did not move with respiration It was uniform and cystic to palpation

Operation revealed a large tumor situated in the midportion of the abdomen it contained approximately 3 quarts of mucoid like fluid The cyst wall was markedly thickened and very vascular and was attached posteriorly to the pancreas and the lower portion of the stomach The stomach was above the tumor and the transverse colon was below The other viscera of the abdomen were negative A trocar was inserted and the fluid was evacuated The sac was drawn out through the abdominal incision as far as possible and the serosa of the sac was then sutured to the peritoneum

The report of macroscopic examination was The wall is firm to palpation and about 0 5 cm in thickness throughout It presents a peritoneal surface in areas covered by rough fibrous bands and contains numerous large blood vessels about 0 5 cm in diameter taking a tortuous course over its surface Other areas are smooth and shiny The inner surface is roughened and pearly gray with several large red hemorrhagic areas Microscopic examination showed a simple pancreatic cyst derived from the duct

Roentgen examination of the colon following the administration of a barium enema (Fig 648) revealed a marked displacement downward of the transverse colon with a crescentic pressure defect extending from just beyond the hepatic flexure to the beginning of the splenic flexure

The diagnosis was of a large intra abdominal mass causing secondary downward displacement of the colon As determined at operation this tumor was a simple cyst of the pancreas

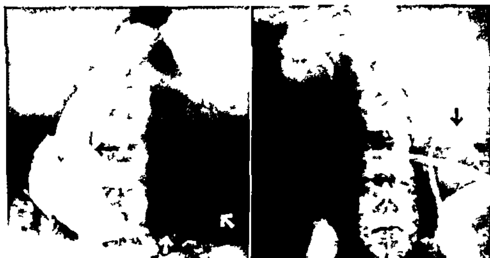


Fig 650 (A *Left*) Pancreatic cyst producing a marked pressure defect on the greater curvature of the stomach with displacement to the right (B *Right*) Same patient as is shown in (A) Note the downward displacement of the left kidney produced by the pancreatic cyst

A pancreatic cyst may displace the stomach to the right

S H female aged 53 During the past 2 years the patient had complained of a burning sensation in the epigastrium about 2 hours before meals This was relieved by soda and

evidence of a mass interposed between the greater curvature of the stomach and the descending colon A duodenal tube was present at the time At another time air was injected into the stomach by way of a duodenal tube and examination was then made in the left lateral, supine position (Fig 646B) This was done as follows With the patient supine and the 14 by 17 casette tangential to the left side the ray was directed through the right side horizontal to the spine The intra abdominal mass indented the posterior wall of the air filled stomach When the stomach was filled with barium, and examination made in the same manner the barium filled stomach was considerably displaced anteriorly, with an increase in the distance between the posterior wall and the spine (Fig 646C) Examination in the erect left lateral position (Fig 646D) also showed an increase in the distance between the spine and the posterior wall of the stomach with evidence of a pressure defect Examination in the left lateral supine position (Fig 646E) without the introduction of barium or air into the stomach shows the soft tissue shadow produced by the tumor Based on this evidence a diagnosis was made of a retrogastric tumor which *in this case* proved to be a carcinoma of the pancreas

Although upward displacement of the stomach and enlargement of the duodenal curve are often indicative of a tumor of the pancreas, these findings may also be produced by a retroperitoneal tumor of different origin

C K, male, aged 46 The patient came to the hospital because of an abdominal mass which had been discovered by his local physician whom he had visited because of a pain in his left thigh He had no abdominal pain or any other symptoms referable to the digestive tract He had lost 7 pounds in the preceding 6 months

Physical examination revealed a large firm, nontender mass occupying the midportion of the abdomen





FIG 649 (A, *Left*) Pancreatic cyst displacing stomach to the right (B, *Right*) Same patient as is shown in (A) Appearance of the resected specimen

At operation there was a large mass occupying the entire central portion of the abdomen The mass was fairly firm, non cystic grayish in color, fixed to the posterior abdominal wall smooth overlying the vertebral column from about the twelfth dorsal vertebra to the fourth lumbar vertebra and anterior to the abdominal aorta Several large fairly firm nodules were present in the region of the bifurcation of the aorta and in both inguinal areas The stomach and intestines showed no abnormalities A biopsy was taken

The pathologic diagnosis was 'The changes are very suggestive of either lymphosarcoma or lymphatic leukemia

Roentgen examination (Fig 647) in the erect position showed upward displacement of the pyloric arm of the stomach and enlargement of the duodenal curve The evidence was that of a retroperitoneal tumor in

and diarrhea associated with left upper quadrant and epigastric cramps of 3 weeks duration Physical examination of the ab domen was negative except for abdominal dis tention and tenderness He recovered from this attack but returned about 6 months later complaining of increasing swelling of the abdomen watery diarrhea and a weight loss of 35 pounds during the preceding year

Abdominal examination revealed ascites requiring paracentesis No tumor cell were found in the ascitic fluid While under ob servation the patient developed considerable discomfort in the left upper quadrant and a mass was felt in that region The mass was 15 cm in diameter fixed had rounded edges felt cystic and was tender and dull to per cussion

At operation a moderate amount of ascitic fluid was present There was a pancreatic cyst which had arisen from the superior sur face of the tail of the pancreas bulged into the area of the lesser sac and was firmly ad herent to the posterior surface of the stomach and the adjacent tissues It was approximately the size of a small grapefruit thin walled and only moderately tense The liver and spleen appeared to be grossly normal There was venous obstruction of the portal system and an increased number of veins Clear fluid ob tained from the cyst was saved for chemical study The lining of the cyst was smooth The cyst was marsupialized Examination of the cyst fluid showed the presence of amylase, protease and lipase

Roentgen examination (Fig 651) in the postero anterior position at 90 degress showed a translucent area of the pars media On fluoroscopic examination there was no evi dence of an intraluminal deformity It was therefore my opinion that the cause was a re trogastric mass which with the patient in the prone position exerted sufficient pressure on the stomach to produce the appearance seen in the roentgenogram As noted at operation the lesion was a pancreatic cyst



FIG 652 A Showing the value of pneumoperitoneum in the visualization of an intra abdominal tumor (cyst of the pancreas)

Pneumoperitoneum may occasionally be of great value in the demonstration of the localization and configuration of an intra abdominal tumor of doubtful origin as is illustrated in Figure 652A In this case physical examination gave no clue as to whether the palpable abdominal tumor was attached to the liver or was independent of this viscus Examination after pneumo peritoneum showed a centrally located fairly sharply delimited mass independent

occasionally by food There was no vomiting or blood in the stool or significant weight loss Physical examination revealed a large, rounded nontender mass about 6 inches in diameter in the left upper quadrant

At operation a large cystic tumor was encountered, involving practically the entire pancreas The cyst was punctured and approximately 3 liters of thick, pale yellow material was aspirated and the mass then dissected from the adherent pancreas The pathologic report was cystadenoma of the pancreas

Roentgen examination (Fig 649A) showed marked displacement of the stomach to the right by the large sharply outlined tumor occupying the left upper quadrant The preoperative opinion was that we were dealing with some form of retroperitoneal tumor the exact nature of which was not determined Renal pathology had been eliminated because of the normal pyelogram Figure 649B shows the appearance of the pancreatic cyst



Fig 651 Cyst of the pancreas, producing a pressure deformity of the body of the stomach

There may be an extreme degree of displacement of the stomach to the right when the pancreatic cyst is very large

P M, female, aged 42 The patient gave a 2 year history of recurring attacks of epigastric pain coming on about 20 minutes after meals and lasting from 20 minutes to 2 hours Three months before her admission to the hospital, the pain had become more severe promptly after meals She vomited frequently and lost 25 pounds during that period She noticed a mass in the upper abdomen Physical examination of the abdomen revealed a firm slightly tender, immobile, nonpulsating mass in the epigastric region extending from the sternum to the umbilicus in the midline and from well over on the left side to the right upper quadrant

At operation a huge pancreatic cyst was found The cyst was opened and quarts of thick, jellylike material were removed The cyst mass was marsupialized to the anterior abdominal wall

The diagnosis was pancreatic cyst (pseudomucinous)

The patient was discharged with a small amount of drainage still present

Roentgen examination (Fig 650A) showed marked displacement of the stomach to the right and a pressure defect on the greater curvature by a large globular mass There was no evidence of any intrinsic lesion of the stomach itself There was marked downward displacement of he left kidney by the mass (Fig 650B) The preoperative roentgen diagnosis was of a large globular tumor occupying mainly the left side of the abdomen and displacing the stomach to the right and the left kidney downward

The patient returned to the hospital 1 year later not only because of the continued drainage through the abdominal sinuses but also because during the preceding month she had developed pain at the site of the sinuses most severe at night

The patient was reoperated on and a pancreatic cyst was found the wall of which was greatly thickened, hard and very friable The lining was thrown into numerous folds and was also hard and irregular in many places The cyst contained foul smelling, white gelatinous material Because of the impossibility of removing the entire cyst the neck was excised as well as a small, hard node in the mesentery of the small bowel

The final diagnosis based on pathologic examination was papillary cyst adenocarcinoma of the pancreas with metastases to the abdominal nodes

A pancreatic cyst when retrogastric may produce a pressure deformity on the posterior wall of the stomach simulating that of an intraluminal lesion

W M male aged 44 The patient was admitted to the hospital complaining of vomiting

intestine with the escape of gas beneath the diaphragm (Kornblith and Otani[5])

PANCREATIC CALCULI

Graff in 1667 is credited with the first reference to pancreatic lithiasis and the condition was recognized by Morgagni in 1765 and Cawley in 1778

Although descriptions of pancreatic lithiasis were at first uncommon roentgen examination since the original report by Assmann[6] has been responsible for the more frequent recognition of the disease and there has been a gradual increase in the number of cases reported In a review of the literature in 1939 Haggard and Kirtley found 65 cases in which the diagnosis was confirmed at operation and 139 nonoperative cases In addition to 80 cases confirmed by autopsy which had been tabulated by Seeger[8] in 1925 Haggard and Kirtley added another 30 with autopsy confirmation

Calcium carbonate is the important constituent of these calculi and since the normal pancreatic secretion contains no calcium carbonate its presence indicates that a profound alteration in its secretion probably the result of infection precedes the formation of these stones

Historically interesting because of its influence on the work of Banting in the development of insulin is the paper by Moses Barron in which he described in great detail the autopsy findings in the case of a calculus of the pancreas which obstructed the duct of Wirsung[9] The findings in the histologic examination of the pancreas revealed changes similar to those which result from experimental ligation of the duct In spite of atrophy of the pancreas with fibrosis the islets of Langerhans remained essentially intact

Pancreatic calculi are best shown in the scout film Because of their localization they may be obscured if barium is introduced into the stomach This is one reason

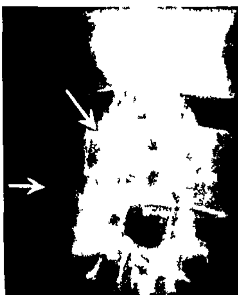




Fig 653 (A *Left*) Pancreatic calculi overlapping the third and fourth lumbar vertebrae (B *Right*) Same patient as is shown in (A) Roentgenogram of the head of the pancreas removed at autopsy showing the anatomic appearance of the pancreatic calculi Note the similarity in the appearance of the specimen itself and the roentgenogram (A)

of the liver and the spleen Operation dis closed this mass to be a cyst of the pan creas The pneumoperitoneal method in this case undoubtedly gave additional aid in the demonstration of the sharp definition of the tumor and its independence of contiguous structures The lateral film is also of value in demonstrating the retroperitoneal loca tion of the tumor (Fig 652B)

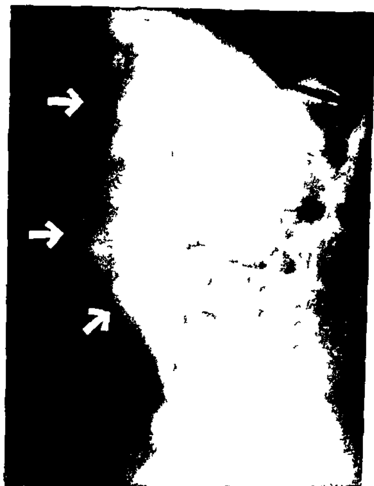


Fig 652 B Same patient as is shown in Figure 652 A Note soft tissue mass produced by the tumor (erect left lateral position)

PANCREATIC FIBROSIS

The relation of pancreatic fibrosis to the development of meconium ileus was first described by *Landsteiner*[4] *on* the basis of the findings at autopsy in a newborn who died of intestinal obstruction This etiologic relationship has since been confirmed by others Because of the diseased condition of the pancreas, there is a serious disturb ance or complete absence of tryptic diges tion The meconium becomes inspissated causing partial or complete obstruction of the small intestine Chronic pulmonary dis ease is commonly found as an associated disorder

Roentgen examination of the intestine *and lungs may thus be helpful in* substantiating the clinical diagnosis of pan creatic fibrosis A rare complication of meconium ileus is perforation of the

abdominal distress This film showed the presence of calculi at about the level of the third and fourth lumbar vertebrae partially overlapping this region (Fig 653 A)

At autopsy there was a massive empyema of the right pleural cavity, perforating the chest wall In addition examination of the pancreas revealed the following The head has a stony hard consistency, and pressure reveals some grating Dissection of the accessory duct reveals a greatly dilated ulcerated duct containing large hard calculi There seems to be an increase of interstitial tissue throughout and the whole organ appears harder to the touch than normal

Microscopic examination revealed a pancreatic fibrosis Figure 653B demonstrates the roentgen appearance of the head of the pancreas as removed at autopsy Note how well the roentgen findings showing the pancreatic calculi at this time correspond to those in the film obtained during life

Although the patient developed attacks of severe abdominal pain while on the ward it is quite likely in view of their localization to the right upper quadrant that they may have been due to the pulmonary pathology with involvement of the right diaphragm particularly since examination of the diaphragm at autopsy showed a few small areas of necrosis and a fibrinopurulent exudate It is therefore difficult to state what role, if any the pancreatic calculi may have played in the production of the abdominal symptoms

Roentgenologically the important features in this case were

1 The location of the calcific shadows in a region which might well correspond anatomically to that of the head of the pancreas

2 An appearance which did not simulate that of a calcified node but for the most part that of a mold of a pancreatic duct

In the following case numerous discrete calculi occurred throughout the pancreas

W F female aged 45 Four weeks before her admission to the hospital the patient developed a persistent nonproductive cough which often kept her awake at night There was considerable vomiting and weight loss of 25 pounds She also complained of pain across the upper part of the back and in both legs Physical examination of the abdomen showed scars in the right upper quadrant the result of two abdominal operations done a number of years ago There was a sinus tract at the site of one of the incisions discharging some fluid

At the first operation 6 years before a mass was found in the pancreas on opening which 5 cc of grayish fluid was evacuated Three years later she was again operated on for a pancreatic abscess and 1 year after that operation showed enlargement of the head of the pancreas due to the formation of a cyst 6 by 4 by 5 cm containing 50 cc of brownish thin odorless fluid rich in protease lipase and amylase The lining of the cavity was irregular grayish yellow It was thought to represent the end stage of a chronic pancreatic abscess A cholecystectomy with choledochostomy was done and drainage and partial marsupialization of the pancreatic cyst

During her stay in the hospital her temperature fluctuated between 99 and 104 Examination of the gastric contents was positive for tubercle bacilli Examination of the chest showed evidence of a tuberculous lesion The patient went rapidly downhill



Fig 655 A Areas of calcification throughout the pancreas

Autopsy revealed fibrocaseous tuberculosis of the lungs with cavitation

The report of examination of the pancreas was The shape of the pancreas is normal Its size is slightly smaller than normal The cut surface of the pancreas is white firm and fibrous There is no recognizable glandular substance The larger pancreatic ducts are markedly dilated and contain numerous greenish brown calculi The largest of these calculi has a diameter of perhaps 4 mm These calculi are especially numerous in the head of the pancreas

Roentgen examination (Fig 654A) showed numerous small calcific areas extending across the spine in the anatomic position of the

among others for taking a preliminary "scout film" of the abdomen at the onset of the roentgen examination particularly in cases that appear to be clinically obscure A lateral film will show the calculi posteriorly placed just anterior to the spine

With the stomach filled with barium, examination in the left lateral position will show the pancreatic calculi to be retrogastric in position If, in addition the pancreas is of increased size, there may be a pressure defect on the posterior wall of the stomach

In most cases, pancreatic calculi are present below the upper margin of the body of calculi were similarly faceted The calculi may frequently be traced from their presence in the head of the pancreas to the right of the spine, across the spine to the left following the course of the body and tail

Illustrative Cases The following case is an example of pancreatic calculi partly outlining the duct

E A, male, aged 45 This patient was well until 8 weeks before his admission to the hospital, when he began having pain of a stabbing nature in the right side of his chest This pain was exaggerated by coughing and deep breathing He complained of marked chills fever, sweats and progressive loss of weight



FIG 654 (A, *Left*) Numerous calcific areas throughout the pancreas (B *Right*) Same patient as is shown in (A) Roentgen appearance of the autopsy specimen

the first lumbar vertebra and above the lower margin of the third lumbar vertebra They vary considerably in size some being minute while others may be from 4 to 5 cm in diameter Calculi may be scattered throughout the pancreas or may form a mold of varying size within the duct The calculi are usually multiple although in rare instances only a single calculus has been found They are of irregular shape and usually strongly resemble the appearance produced by calcified lymph nodes Such were the findings in the cases reported by Wolf and Tietze[10] and Marshall[11] Rarely they may be faceted[1]

Gillies was able to find a record of only one other case besides his own in which the

Physical examination as well as roentgen study disclosed a suppurative pneumonitis involving the right lower lobe of the lung While on the ward the patient developed severe right upper quadrant pain The right abdominal wall was markedly spastic, board like in rigidity with right upper quadrant tenderness The left side of the abdomen was normal The abdominal complaints subsided by the next day and examination at that time revealed the abdomen to be soft and without tenderness Cough and fever persisted however and several weeks later he had another episode of acute abdominal pain Thoracotomy and drainage of the right lower lobe was done

Roentgen examination was as follows In addition to roentgen study of the chest which demonstrated the pulmonary involvement a scout film had been taken at the time of his

common duct at its entrance into the duodenum Numerous calculi from 5 to 1 cm in diameter and having jagged edges are found in the head of the pancreas The rest of the pancreas is atrophied about a very widely dilated main duct of the pancreas The diameter of this duct is approximately 2½ cm There are numerous calculi present also in the tail of the pancreas

Microscopic examination revealed carcinoma of the pancreas

The inner border of the second portion of the duodenum showed a deformity of contour with an atypical inverted figure 3 effect, as well as irregularly outlined translucent areas, which suggested that a tumor of the head of the pancreas was present which had invaded the lumen of the duodenum Although a carcinoma of the head of the pancreas was found at autopsy the duodenum showed *no evidence* of intrinsic disease The translucent areas may therefore be considered as having been



Fig 656 Calcified areas throughout the pancreas Examination in the erect left lateral position

Roentgen examination (Fig 655A) showed areas of calcification in the anatomic position of the pancreas A number of the calcific areas were present to the right of the lower portion of the first lumbar vertebra and of the second lumbar vertebra evidently representing the head of the pancreas A number of calcifications were also present *in the left* upper quadrant in the position of the tail of the pancreas

In the examination of the stomach and duodenum (Fig 655B) some of the calculi were noted within the confines *of the duodenal curve* and from there the calcifications could be traced obliquely upward and to the left

produced by pressure of the enlarged head of the pancreas

Figure 655C is a reproduction of the roentgenogram of the pancreas removed at autopsy and shows the irregularly outlined calcifications

The value of the lateral film in the demonstration of pancreatic calculi may be seen in Figure 656

The occurrence of calculi of extremely varied size diffusely spread throughout the entire pancreas is well illustrated by the following case

pancreas Most of the areas of calcification were in the region of the head of the pancreas to the right of the second and third



FIG 655 B Same patient as is shown in Figure 655 A Appearance of the duodenal curve Note the inverted 3 at the inner border of the second portion of the duodenum produced by carcinoma of the head of the pancreas Note the position of calcific areas in the pancreas

The diagnosis was calcification of the pancreas

At autopsy the pancreas was removed and radiographed (Fig 654B) This shows areas of calcification throughout the pancreas

The next case illustrates the findings in a case of pancreatic lithiasis and carcinoma of the head of the pancreas

G R female aged 57 During the preceding 3 years the patient had complained of pain in the right upper quadrant with radiation to the right shoulder blade made worse after meals lasting from 15 minutes to 1 hour and relieved by induced vomiting She had lost 15 pounds in weight She had one attack of diarrhea lasting 2 weeks

During the preceding 2 months she had become jaundiced Physical examination revealed deep jaundice The liver was palpable three fingers breadth below the right costal margin, with a suggestion of a nodular edge A mass was palpable in the right upper quadrant The fingers and toes were clubbed

At operation there was dilatation of the gallbladder and extrahepatic ducts The pancreas was enlarged thickened and nodular throughout

The operative diagnosis was chronic pancreatitis and pancreatic lithiasis A cholecystgastrostomy was done



FIG 655 C Same patient as is shown in Figure 655 A and B Roentgen appearance of the autopsy specimen of the pancreas showing the calcifications

lumbar vertebrae A number of calcifications were also noted in the area corresponding to the body and tail of the pancreas Barium was present in the colon at the time of this examination

The autopsy findings were as follows The head of the pancreas is thickened and hard to palpation It is dark brown to dark green in color It encircles and produces complete stenosis of t e main pancreatic duct and the

common duct at its entrance into the duodenum Numerous calculi from 5 to 1 cm in diameter and having jagged edges are found in the head of the pancreas The rest of the pancreas is atrophied about a very widely dilated main duct of the pancreas The diameter of this duct is approximately 2½ cm There are numerous calculi present also in the tail of the pancreas

Microscopic examination revealed carcinoma of the pancreas

The inner border of the second portion of the duodenum showed a deformity of contour with an atypical inverted figure 3 effect, as well as irregularly outlined translucent areas which suggested that a tumor of the head of the pancreas was present which had invaded the lumen of the duodenum Although a carcinoma of the head of the pancreas was found at autopsy the duodenum showed no evidence of intrinsic disease The translucent areas may therefore be considered as having been

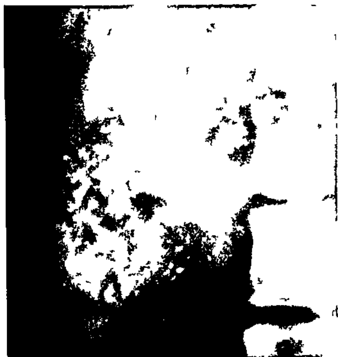


Fig 656 Calcified areas throughout the pancreas Examination in the erect left lateral position

Roentgen examination (Fig 655A) showed areas of calcification in the anatomic position of the pancreas A number of the calcific areas were present to the right of the lower portion of the first lumbar vertebra and of the second lumbar vertebra evidently representing the head of the pancreas A number of calcifications were also present in the left upper quadrant in the position of the tail of the pancreas

In the examination of the stomach and duodenum (Fig 655B) some of the calculi were noted within the confines of the duodenal curve and from there the calcifications could be traced obliquely upward and to the left

produced by pressure of the enlarged head of the pancreas

Figure 655C is a reproduction of the roentgenogram of the pancreas removed at autopsy and shows the irregularly outlined calcifications

The value of the lateral film in the demonstration of pancreatic calculi may be seen in Figure 656

The occurrence of calculi of extremely varied size diffusely spread throughout the entire pancreas is well illustrated by the following case



Fig 657 (A *Top*) Shows irregularly outlined opaque areas throughout the head and body of the pancreas Note the unusual position and configuration of the stomach which was found to be normal at autopsy The defect of the pyloric portion of the stomach was apparently produced by pressure of the head of the pancreas (B *Bottom*) Same patient as is shown in (A) Roentgen appearance of the autopsy specimen Note the evidence of calcification throughout the pancreas



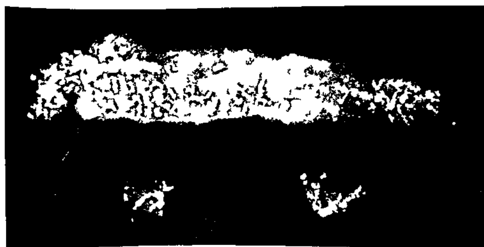


FIG 658 (A *Top*) Note the calcifications of the pancreas Most of those seen are in the head of the pancreas Note also the pressure on the pylorus produced by the head of the pancreas (B *Bottom*) Same patient as is shown in (C) Roentgen appearance of these calcifications in the resected specimen

M C, male, aged 60 The patient stated that 2 years prior to his admission to the hospital he had had mild abdominal and epigastric pain which was worse after eating but not associated with vomiting The pain gradually became worse and finally was constant day and night He then complained of vomiting mainly after meals About 6 months before his admission there was a period of 3 months when he had from 10 to 12 yellow, *"bulky" stools daily He lost 40 pounds during the 2 years of his illness, of which 20* were lost in the preceding 2 months Physical examination revealed evidence of pulmonary tuberculosis

Examination of the abdomen was essentially negative

are rough with numerous small spicules Around these there is in the lumen a thin, purulent appearing exudate The pancreas in general has a normal contour and size Numerous gravel like particles can be palpated throughout the organ even outside the main duct On section, the lobular architecture is distorted by broad, white fibrous strands and by autolytic changes It is gray and soft No areas of fat necrosis or of fibrous adhesions are seen Several nodes at the head of the pancreas contain anthracotic pigment As stated previously, the splenic artery, portal vein and its sources are normal"

The microscopic examination was reported as follows "Both sections have been decalcified The first section had been taken from the



FIG 659 (A, *Left*) Case of pancreatic abscess Areas of calcification throughout the pancreas (B *Right*) Same patient as is shown in (A) Examination in the erect position showing the abscess with fluid level

At autopsy there was fibrocaseous tuberculosis of the right upper and right middle lobe with cavity formation and bronchogenic dissemination to all the lobes The gastro intestinal tract from the pharynx down to the terminal ileum was normal Approximately 2 feet proximal to the cecum there was an ulcer of the ileum There was also a superficial ulcer 4 cm above the anus Examination of the gallbladder and of the biliary passages was negative

Examination of the pancreas showed the following "The pancreatic duct opens into the papilla of Vater independently of the common bile duct and below it It is markedly distended throughout the length of the organ being approximately 8 mm in diameter on the average It is filled with numerous stones in its entire length stones which vary in length from 1 5 cm to the size of coarse sand These are light yellow gray and their surfaces

head of the pancreas close to the papilla of Vater Most of the mucosa of the common bile duct has been nipped away, that which remains is columnar and normal The muscular wall is well developed in evidence of its proximity to the papilla It is not dilated or inflamed The pancreatic parenchyma has been almost entirely replaced by dense acellular collagenous tissue A rare acinus is present, but a greater number of fairly well preserved islets are seen in the head and in the section taken from the tail *The ducts are* greatly distended and their mucosa desquamated Within the lumen there is frequently an exudate of polymorphonuclear leukocytes and a variable proportion of lymphocytes and mononuclears as well as granular debris The walls and adjacent structures too are infiltrated with a moderate number of inflammatory cell The smaller arteries show mild to moderate hyalinization of their walls"

The final diagnosis was stated as "pancreatic lithiasis fibrosis and atrophy of the pancreas and chronic inflammation of the ducts

Roentgen examination (Fig 657A) revealed no evidence of any organic pathology of the stomach or duodenum In the prone position however, the stomach showed considerable abnormality of its position, to be explained in part at least, on the basis of exaggerated mobility There is also evidence of a pressure defect of the distal portion of the stomach In addition, there are numerous small calcific areas in the region of the pressure deformity of the stomach as well as others to the left of the midline These were interpreted as due to calculi throughout the head and body of the pancreas It was my own opinion that the pressure deformity was produced by an enlarged head of the pancreas

As noted in the autopsy description the stomach was found to be entirely normal Of considerable interest is the fact that the pathologist considered both the contour as well as the size of the pancreas to be normal

Figure 657B shows the roentgen appearance of the pancreas removed at autopsy

Diffuse calcification of the pancreas in a patient in whom an almost total pancreatectomy was done is illustrated by the following case

A S female aged 28 The patient gave a 1½ year history of recurring attacks of stabbing epigastric pain radiating to the back and across the shoulders The attacks usually occurred about twice a month each episode lasting about a week They might occur at any time of the day or night and were accompanied by blood free vomiting There were no chills fever or jaundice There was occasional diarrhea

Physical examination was essentially negative except for tenderness in the right upper quadrant

At operation the pancreas was found to be diffusely calcified from head to tail The head of the pancreas was from two and one half to three times the normal size was of cystic appearance and feel with multiple calcified areas An almost total pancreatectomy was done

The findings on pathologic examination were The pancreas is pinkish white measures 13 cm x 2 5 cm The surface is irregular and the entire pancreas contains countless numbers of small white calcific deposits These are largest in the pancreatic duct, as seen by section but are spread throughout all parts of the pancreas as well They vary from pinhead size to 2 mm in diameter There are two additional pieces of pancreas each measuring 2 cm x 3 cm x 2 cm and having a similar composition The diagnosis was pancreatic lithiasis fibrosis of the pancreas

Roentgen examination (Fig 658A) shows areas of calcification throughout the pancreas Most of the calculi observed are in the head



Fig 659 C Same patient as is shown in Figure 659 A and B Examination in the left lateral decubitus position showing a gas capped fluid level within the pancreatic abscess

of the pancreas There is a deformity of the pyloric portion of the stomach evidently due to pressure of the enlarged head of the pancreas Figure 658B represents the roentgen appearance of the resected pancreas showing the widespread calcifications

An abscess of pancreatic origin associated with calculi is illustrated by the following case

F J male aged 34 The patient gave a 10 year history of recurrent attacks of severe upper abdominal pain radiating to the back and left upper quadrant lasting from 1 to 3 days but not requiring hospitalization The attacks ultimately became incapacitating, and

while in the army he had a cholecystectomy without relief Three weeks before admission to the hospital he developed a very severe attack of epigastric pain with vomiting and swelling of the abdomen and high fever Physical examination of the abdomen revealed uniform distention, generalized tenderness and rebound tenderness most marked in the epigastrium

At operation a huge well walled off abscess cavity was found containing about 2,000 cc of chocolate colored fluid and pus Examination of the fluid from the abscess cavity revealed amylase 365 mg

Roentgen examination had revealed the following There was evidence of calcification of the pancreas (Fig 659A) *In addition* examination in the erect position showed a mass occupying the mid upper area of the abdomen with a fluid level capped by gas (Fig 659B) In the left lateral decubitus *position the* fluid level within the mass capped by gas was clearly visible (Fig 659C) A diagnosis of pancreatic abscess was made for the following reasons Because of the presence of calculi in the pancreas it was assumed that the patient had an associated pancreatitis The cavity with the fluid level was considered to be an abscess Its location was such that it might very well have originated from a diseased pancreas Not only was an abscess found at operation but examination of the fluid content revealed 365 mg of amylase This latter finding added corroboration to the diagnosis of pancreatic abscess

About 1 year later the patient was reoperated Several stones were removed from the body and tail of the pancreas and the head was resected The isolated stones as well as the resected specimen were radiographed (Fig 659D) Note in particular the remaining calculi within the head of the pancreas *The preoperative roentgen diagnosis of pancreatic lithiasis* was therefore found to be fully confirmed



FIG 659 D Same patient as is shown in *Figure 659 A B and C Roentgen appearance* of stones removed from the pancreas as well as those in the resected head

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Bibliographic Index

Subject Index

All numerals in boldface type refer to Figures used on the de ignated pages of the text